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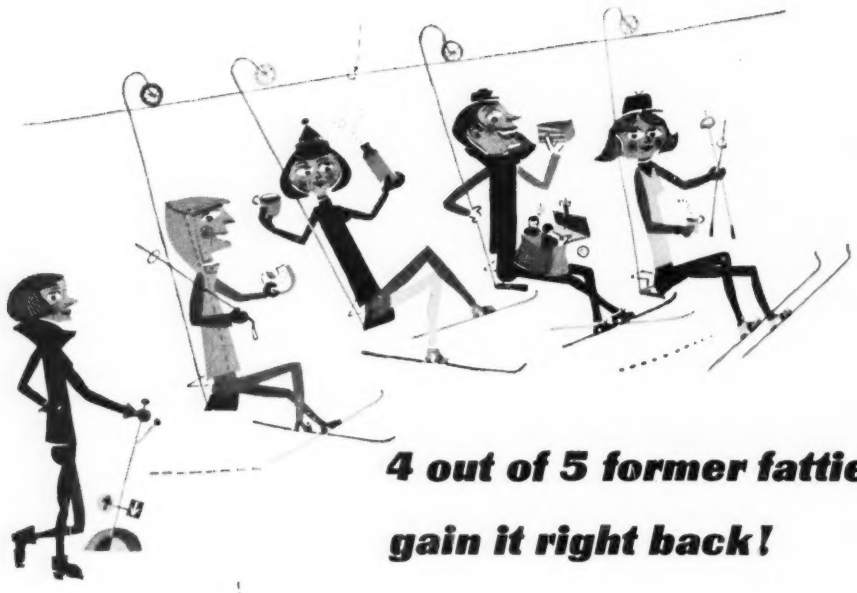

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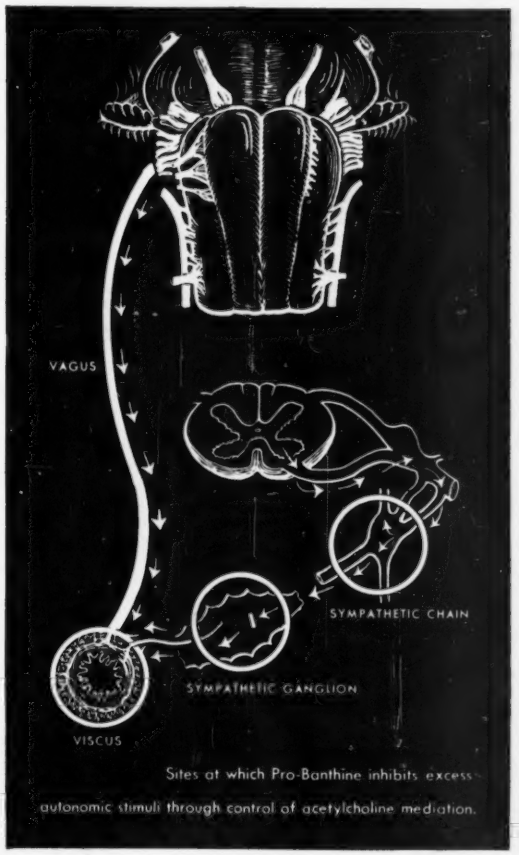

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VOL. 22, No 6 DECEMBER, 1954

THE American Journal OF Gastroenterology

(FORMERLY THE REVIEW OF GASTROENTEROLOGY)

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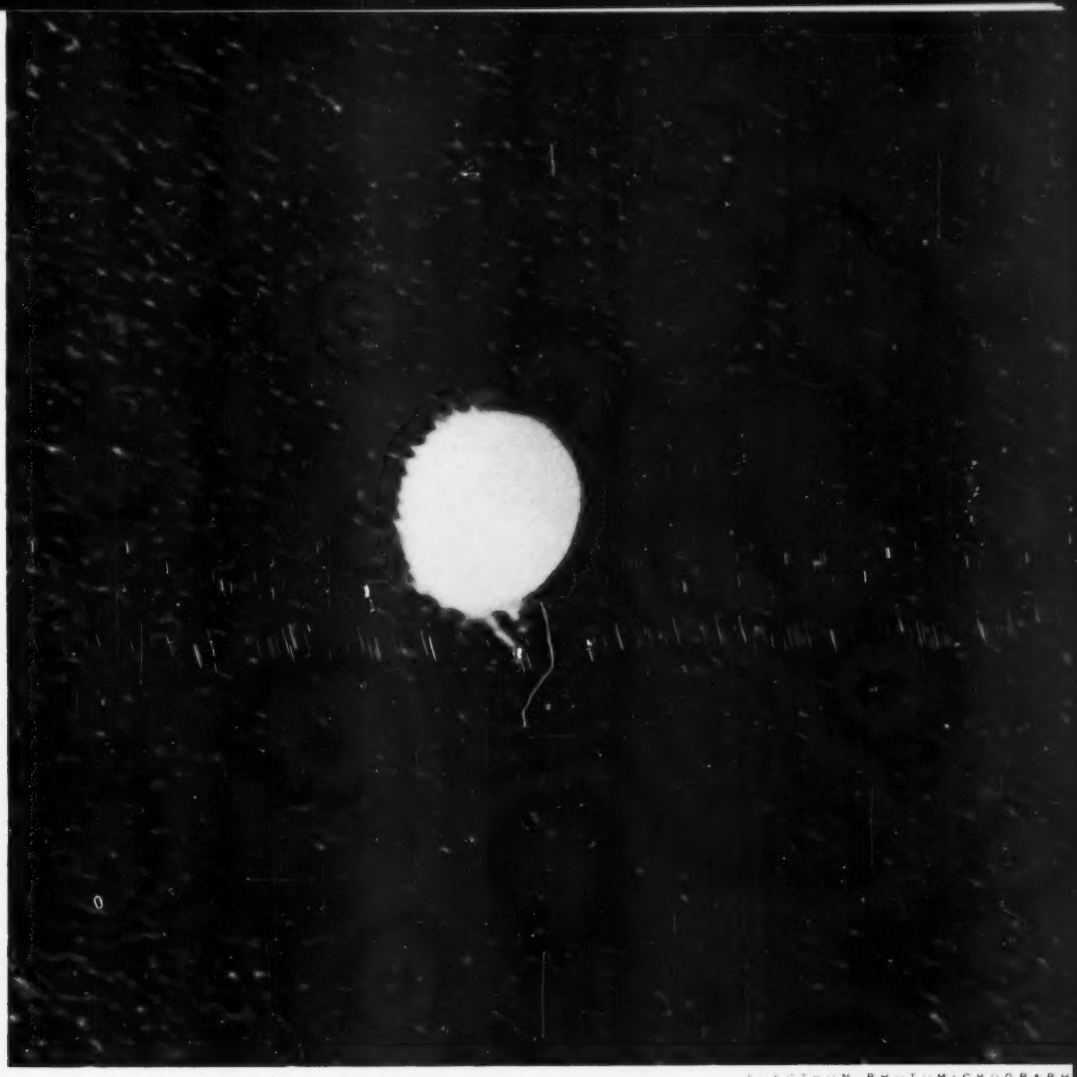

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NEWER DRUGS FOR THE MANAGEMENT OF PEPTIC ULCER*

AN ANALYSIS

MURREL H. KAPLAN, M.D., F.A.C.G.†

New Orleans, La.

In 1944, while in Italy with the United States Armed Forces, there was an opportunity to observe patients with peptic ulcer from an entirely different aspect than previously afforded. Patients whose conditions had been diagnosed as peptic ulcer were divided into two groups as they were received from both forward area and nearby hospitals. The first group was treated with a modified Sippy regimen (purees with frequent mild feedings, antacids, and antispasmodics); the second group was given a bland diet, mild sedation, and daily interviews, which psychiatrists are wont to term "mental catharsis". After a period of a few weeks, roentgenologic studies were checked; the second group of patients showed the greater incidence of improvement. From this experience, I was prompted to relegate drug therapy to a secondary role in the management of peptic ulcer, and to regard newer medications with some degree of skepticism.

CRITERIA OF RESPONSE

Certain criteria of response must be considered in evaluating any drug proposed for the treatment of peptic ulcer. The three criteria suggested by Todd[13] are: (1) Immediate symptomatic response, (2) Influence on the healing of the lesion, and (3) The ability of the drug to change the natural course of the disease. The immediate symptomatic response infers the rapidity with which symptoms disappear or fail to recur in terms of hours after initial administration of the drug, and the number of days following which the patient is asymptomatic. Roentgenologic studies serve to demonstrate the influence of a drug upon healing of the lesion. Superficial lesions may heal in a few days while deep lesions may require from one to twenty weeks for healing. The

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†Department of Medicine, Louisiana State University, Department of Gastroenterology, Touro Infirmary.

accepted average is about 40 days. Gastroscopic examination will reveal the drug's effect upon the healing of gastric ulcers. The ability of a drug to alter the course of the disease by reducing the percentage of complications and the frequency of recurrence is a criterion that necessarily includes patients undergoing surgery either as an emergency or as an elective procedure. Finally I may add a fourth criterion which is the ability of a drug to be tolerated without too many or too severe reactions. It may be mentioned that 606 trials were attempted by Ehrlich before one therapeutic measure was found that was not disastrous to the patient at the time it was destroying the spirochete of syphilis.

One must be aware of the following pitfalls when interpreting the results of therapy for peptic ulcer: (1) Spontaneous remissions, (2) Psychological effects, (3) Relationship of severity of symptoms and of demonstrated pathologic change to relief from medication, and (4) Variations in interpretations of fluoroscopic and x-ray examinations. Spontaneous remission is the so-called natural course of the illness. Intermittent remissions, however, without diet or medication, also seasonal exacerbations, remain most baffling conditions. The psychological effects, other than planned psychotherapy, are to be considered. Hospitalization or a change of environment may effect a so-called "cure". Placebos have proved effective in the relief of pain and symptoms[13], although they may not heal the condition[10]. This brings to mind the enthusiastic reports of the success of Larostidin injections before similar results were obtained by the use of distilled water.

It is not uncommon to observe a patient with a typical history of a penetrating ulcer but without demonstrable lesions on the x-ray film. On the other hand, there are cases of mild dyspepsia with craters, shallow or deep, active or healed, demonstrated on films. Therefore, the evaluation and relationship of the severity of symptoms and demonstrated organic pathology following medication is on an individual basis and cannot be correlated with x-ray findings.

It is my belief that not only the extent of tissue destroyed but also the personality of the patient determine the severity of complaints and response to medication. There exists wide variation of opinion among roentgenologists and gastroenterologists who "take" and interpret their own films. Duodenal irritabilities, spasm, and superficial ulceration are the sources of controversy while the chronic ulcer crater provokes no discussion. Frequently, and possibly correctly, conclusions are "read into" our fluoroscopic and x-ray examinations.

Development of Peptic Ulcer Therapy

Although Hippocrates (460 B.C.), described the symptoms of peptic ulcer, it was Celsus in the first century A.D. who recognized the importance of acid neutralization in the relief of abdominal distress. Credit is given to Pemberton in the 18th century for suggesting frequent feedings and alkaline medication in these cases. In 1856, Cruveilhier became known as the "great advocate of the

milk diet", as cited by Sandweiss[10]. In the late 19th century, German investigators stressed starvation and alkaline waters, at the same time minimizing the value of neutralization of acids. Notable amongst these were Ewald and Boas whose pupil, Einhorn, popularized this type of therapy in the United States.

B. W. Sippy, in 1912, reapplied the principles of frequent feedings and alkalis especially sodium bicarbonate[11]. In 1918, Crohn, as cited by Sandweiss[10] demonstrated the so-called reactive effects of sodium bicarbonate on gastric acidity and in 1923 Hardt and Rivers reported cases of alkalosis resulting from its use[7]. This sounded the death knell of "cooking soda" as an alkali and stimulated the research of nonabsorbable antacids. Immediately other forms of therapy were suggested; i.e., resins, mucins, hormones, protein hydrolysates, detergents, and the most recent group of blocking agents, the anticholinergics.

Pathologic Physiology

Years of research in the physiology laboratory have resulted in an unanimous opinion that whatever may be the causes of peptic ulcer, the condition was aggravated by acid pepsin digestion. Recent experiments by Ruffin and coworkers[14] suggested that abnormal motility is the fundamental mechanism that produces ulcer pain. The approach therefore to the so-called "cure" of peptic ulcer must depend upon: (1) Suppression of the cells of the stomach by which acid pepsin production is depressed, (2) Neutralization of the secreted acid pepsin factors; (3) Alteration of the abnormal motility of the gastroduodenal segment, and (4) Increasing the resistance of the cells of the stomach and/or duodenum to the effects of the acid pepsin factors.

Rise of the Anticholinergics

Shortly after Dragstedt, in 1945[4], revived the procedure of vagotomy for peptic ulcer others began to seek a drug which would have the same physiologic effect as had the severance of the vagal nerves. In 1950, Grimson and associates[5] reported the use of the autonomic blocking agent, beta-diethylaminoethyl-xanthen-9-carboxylate methrobromine, Banthine. The drug was received with enthusiasm for it seemed to be the realization of the dream of "medical vagotomy", that of effecting a reduction in gastric sercretion and an inhibition of gastric motility. As further studies were made, the inhibitory activity of Banthine was more constant and predictable than the depressant action on gastric secretion following vagotomy. Bachrach and his associates in California[1], among other workers, have recently demonstrated that anticholinergics fail to reduce the acid content of the human stomach. The humoral theory satisfactorily explains these findings. Concerning this concept the following statement is quoted in part from an editorial in the *Journal of the American Medical Association*[3]: "In response to emotional and systemic factors, the cells of the anterior hypothalamus secrete a humoral substance that stimulates the pituitary

gland to release corticoids (as cortisone) from the adrenal cortex, and these in turn stimulate the gastric glands to an increased production of pepsin and hydrochloric acid".

Value and Applicability of the Anticholinergics

Banthine and similar blocking agents (Prantal, Centrine, Antrenyl, etc.) soon became popularly used. In the literature there appeared glowing reports of the rapidity with which (1) pain of peptic ulcer was relieved, and (2) x-ray findings demonstrated evidence of healing[2,6,8]. Among investigators who appreciated the limitations of these agents were McHardy, Browne and their associates[9] who, in 1951, published their results and stressed the fact that while Banthine relieved the pain of peptic ulcer, it was not the final answer to the therapy of this condition.

The anticholinergic drugs have been accepted as effecting relief of the pain of peptic ulcer, thereby fulfilling the criterion of symptomatic relief as above outlined, and have been found to be more quickly effective than atropine and belladonna[12]. A report of 18 cases indicated that the healing time, as shown by x-ray, was dramatically shortened following the use of Banthine[6] Other observations fail to confirm this finding and the general opinion is that there are no accurate data available as to the healing time of duodenal ulcer following the use of anticholinergics. The blocking agents have failed to alter the natural course of the disease and this finding may be summed-up in the following statement by Ruffin[14]. "These agents have been disappointing in the long term management of ulcer since recurrences are not prevented and the incidence of complications or the need for surgery are not significantly altered by their prolonged administration".

Side-effects of the Anticholinergics

The side-effects of Banthine and the related group of drugs include urinary retention, mydriasis, xerostomia, constipation and abdominal distention, headaches, dizziness and lassitude. It is very disturbing for a patient, already besieged with anxiety, to experience additional difficulty in emptying his urinary bladder. The drugs may cause blurring of vision which increases apprehension in a patient for whom every psychotherapeutic means is being employed to induce calm. Dryness of the mouth also is disconcerting, and constipation and abdominal distention may cause distress in some ulcer patients who are already figuratively "tied in knots".

One may ask what of the complicated cases of peptic ulcer, if these newer drugs are not absolutely necessary in the management of the uncomplicated case? The answer to the question concerning any and all complicated cases of peptic ulcer may be found in the following general statement: "In any patient in whom abdominal surgery is contemplated, these drugs should not be used

during the immediate preoperative period because of the dilatation and paralysis of the stomach and small intestine"[14]. Hemorrhage, penetration, perforation, and obstruction are potential surgical problems. At the present time there are no available statistics as to the use of the anticholinergics in the incidence of these complications. It may be that the gastroenterologist is not eager to witness the reactions of the surgeon who must operate on a "banthinized" patient. It is generally accepted that the anticholinergic drugs are contraindicated in cases of perforation and of cicatricial obstruction.

Conclusion

It has been my experience and that of my associates that the anticholinergic drugs are useful as an adjunct in the management of peptic ulcer but have few advantages over the standard belladonna drugs. The untoward reactions have been sufficiently distressing to cause their usage to be limited to the uncomplicated cases that do not readily respond to the previously accepted therapeutic agents.

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FURTHER STUDIES ON PROLAPSE OF THE GASTRIC MUCOSA INTO THE DUODENUM*

MAURICE FELDMAN, M.D.

Baltimore, Md.

In previous publications[23-26] on prolapse of the gastric mucosa into the duodenal bulb, we attempted to clarify the clinical manifestations of this entity. The literature was reviewed up to 1952 and the pertinent statistical data was assembled. Correlations were then made between the clinical and surgical proven cases, in the belief that a clinical syndrome could be established for this condition. The present study was made to bring the additional clinical material and the literature up to date.

Up to 1952, there were 87 publications on prolapse of the gastric mucosa, with a report of 742 cases listed in the literature, of which 131 cases were surgically explored. Since then, there have been 59 additional references to this condition, with a report of 324 cases, making a total of 146 publications and 1,066 cases recorded up to January 1954. Of these, 78 were surgically proven, making a total of 209 surgically explored cases reported to date.

The incidence of prolapse of the gastric mucosa has been established by numerous authors. Although a survey of the earlier literature revealed an extremely low incidence of this condition, more recently, most authorities have found a higher incidence in the general population and a still greater incidence among patients complaining of gastrointestinal disturbances. For instance, among 371 consecutive gastrointestinal cases, we found an incidence of 14 per cent. It is generally conceded, however, that the incidence in the general population is probably around 10 per cent. The reported incidences are shown in Table I.

The surgical incidence has not been fully established. Since the condition is essentially a medical problem, the surgical incidence should be very low. Appleby noted 7 instances of prolapse of the gastric mucosa among his surgical cases, or an incidence of 1.1 per cent. Among the 1,066 recorded cases, 209 were surgically explored, or an incidence of 19.6 per cent. The latter incidence is certainly not the true surgical incidence, since only the severe type and the intractable cases necessitated surgical exploration, and these were reported with greater frequency.

The autopsy incidence has not been established. In 1,500 autopsies studied previously, not a single instance of prolapse of the gastric mucosa was noted, since then, however, our pathologists have been on the lookout for the con-

*Read before the Gastroenterological Section of the District of Columbia Medical Society, Washington, D. C. on April 12, 1954.

dition and they recently were able to find one autopsy case in which the gastric mucosa was prolapsed into the second portion of the duodenum. This condition has been confirmed at autopsy in many instances. More recently Stiller reported 4 cases of prolapse of the gastric mucosa proved by autopsy findings.

The basic abnormality in prolapse of the gastric mucosa is an excessive mobility of the mucosa upon the muscularis. The amount of mucosal redundancy associated with excessive peristaltic action no doubt accounts for the pro-

TABLE I

ROENTGENOLOGIC INCIDENCE OF PROLAPSE OF THE GASTRIC MUCOSA

	Cases X-rayed	Prolapse of Gastric Mucosa Cases	Per cent
Morris	2,184	133	6.13
Paltriniere	1,043	17	1.6
New	1,700		2.9
Melamed et al.	2,404	150	6.2
Rappaport et al.	1,000	155	15.5
Bartels and Eltorm	240	44	18.3
Weens	297	23	7.7
Johnson	1,593	117	7.3
Spencer et al.	300	9	3.0
Feldman et al. (Collected series)	20,464	363	1.8
Feldman and Myers	371	52	14.0

pelling force and the degree of prolapse. Whether or not gastritis is a primary or secondary condition has not been clearly established. Prolapsing gastric mucosa is associated with varying degrees of gastritis in almost 50 per cent of cases. King had noted that in 100 per cent of alcoholic patients with hypertrophic gastritis, there was evidence of a prolapse of the gastric mucosa. Lichtstein and Asher observed that acute symptoms may be precipitated by excessive alcohol drinking. This is perhaps due to the effect of the alcohol on the gastritis. Many authors have theorized on the etiology of the gastritis. It is claimed that

the chronic inflammation may be due to traumatization and irritation resulting from the to and fro movements of the prolapsed mucosa. Others are of the opinion that there is an inherent primary condition of the antral mucosa. Some believe that narrowing of the pyloric outlet precedes the hypertrophic gastritis, and that hyperperistalsis occurs secondarily. Another possible secondary cause is a primary pyloroduodenal ulceration.

Hyperperistalsis is found in the majority of cases. It may be due to, (1) increased effort to overcome the ball and valve effect or to a narrowed pylorus, (2) foreign body effect, (3) stimulus by an antral gastritis, and (4) secondary to an associated duodenal ulcer.

Prolapse of the gastric mucosa occurs more often between the 3rd and 6th decades of life, and predominantly in the male sex. In 432 cases, there were 309 or 71.5 per cent males, and 123 or 28.5 per cent females.

The clinical picture of prolapse of the gastric mucosa is that of a pyloroduodenal disturbance. It is characterized by periodic attacks of pain, abdominal discomfort, indigestion, nausea and vomiting, bleeding, (hematemesis, melena or tarry stools), gas, belching, pyrosis, and late signs of anemia, weakness and loss of weight. Like other intermittent affections, prolapse of the gastric mucosa may not produce any symptoms whatsoever, or it may occur as an incidental finding in patients with pathology unrelated to the stomach or duodenum. The epigastric pain varies in intensity, and is often of a cramp-like character. The pain is commonly produced by eating, and is more pronounced following solid foods and fats. In the uncomplicated case, there is usually an absence of night pain and no relief from food or antacids. The symptoms may be aggravated when the patient assumes the recumbent position. Patients are often afraid to eat because of pain and vomiting. In the absence of an associated duodenal ulcer, food aggravation rather than relief is more or less characteristic of this condition. Usually there are asymptomatic periods and also temporary relief following the administration of an antispasmodic or anticholinergic drug.

Physical examination in the milder cases shows nothing abnormal. Some degree of epigastric sensitiveness or tenderness, however, may be elicited on deep pressure in many instances. A palpable mass is not often felt. In those cases with extensive prolapse of the mucosa, it may be possible to palpate a doughy mass in some cases.

Gastric analysis yields acid values ranging from an extreme low to a moderately high acidity. In most instances the acid values are normal.

Gastroscopic examination aids in the differential diagnosis. Gastroscopy usually reveals a redundancy and evidence of a gastritis of the antral mucosa.

The essential pathologic findings are redundant gastric mucosa, loosened attachment of the antral mucosa from the submucosa and gastritis. The antral

mucosa show large edematous rugae, with disorientation and rotation of the folds. The vast majority of cases show some degree of hypertrophic gastritis, but in a few instances an atrophic gastritis is observed. Allende and DeArteaga reported a case associated with a cystic gastritis. In some instances the pyloric musculature is thickened with resultant narrowing of the pyloric canal. The pyloric outlet is more often widened, owing to the distention produced by the mass of prolapsed mucosa. The redundant mucosa shows evidence of congestion, erosion, superficial ulceration, and at times pseudopolyp formation, polypoid degeneration and rarely benign or malignant neoplasms.

The roentgenologic criteria for the diagnosis of prolapse of the gastric mucosa is pathognomonic. Most clinicians, surgeons and roentgenologists now concede that prolapse of the gastric mucosa into the duodenum is a clinical entity. The roentgenologic data has been discussed in detail in previous publications and emphasis on certain characteristics of the condition will be presented here. It must be pointed out that the degree of prolapse often varies with the position in which the roentgenograms are made, as well as to peristaltic activity. There is often a difference in the findings between the fluoroscopic and roentgenographic views. Although the prolapse can be demonstrated in all positions, it is best observed in the prone, and especially in the right oblique position. During the fluoroscopic examination the herniated gastric mucosa can sometimes be reduced back into the stomach by manipulating pressure, following relaxation of the pyloric sphincter, during the phase of lessened peristaltic activity, or after the use of an anticholinergic drug. When the prolapse is temporarily reduced, the redundant mucosa piles up into the pyloric antrum, where the rugae are shown to be disorientated, assuming a circular, oblique, transverse or criss-cross pattern, instead of the usual longitudinal one.

It must be emphasized that minor changes in the base of the duodenal bulb may be observed in normal individuals and these cases are usually excluded by most roentgenologists. Basing his findings on freshly resected specimens, Schröder pointed out that the umbrella or mushroom filling defect in the bulb may be produced by other conditions than prolapse, such as contraction of the pyloric sphincter, and swollen edematous mucosa. Although these findings must be excluded, yet, they do not represent the true picture of prolapse of the gastric mucosa into the duodenal bulb, since in the latter instance, the redundant prolapsed mucosa presents a to and fro movement from the stomach into the duodenum. The degree of prolapse of the gastric mucosa may be classified as, slight, moderate and marked. In cases with extensive prolapse, the mucosal mass may herniate into the second or third portions and rarely into the fourth portion of the duodenum.

The most common associated conditions noted are gastritis and duodenal ulceration. Gastritis occurs in from 40 to 50 per cent of cases. The incidence of duodenal ulcer is greater among cases with prolapse of the gastric mucosa than

is found otherwise. In 705 collected cases, there were 127 with duodenal ulcer, an incidence of 18 per cent. Attention must also be directed to the fact that at times the prolapsed gastric mucosa may mask a duodenal ulcer filling defect. Pseudopolyps or polypoid degeneration has been reported in only a few instances. The polypoid changes are essentially the pseudopolyp variety secondary to the gastritis. The association of a benign tumor or carcinoma of the stomach with prolapse of the mucosa is comparatively rare.

In the differential diagnosis, the following conditions must be excluded: peptic ulcer, duodenitis, antral spasm, hypertrophic pyloric stenosis, prolapsing benign tumors, invagination of the pylorus into the duodenum, and carcinoma. The symptoms of prolapse of the gastric mucosa often simulate an atypical duodenal ulcer syndrome. In most instances, however, it differs in certain characteristics, such as, aggravation of pain by food, failure of relief from alkalis, no night pain, its occurrence in the older age group, the usual absence of hyperacidity, a greater tendency to nausea and vomiting, and higher incidence of gas and abdominal fullness.

The association of a prolapsing gastric tumor with prolapsing antral mucosa should reveal a characteristic roentgen picture of a triple filling defect, showing a polyp type defect in the duodenal bulb, a pyloric defect produced by the hypertrophied antral mucosa, and a contour defect resulting from the attachment of the pedunculated tumor. It must be emphasized that it is highly important to observe the appearance of the pyloric canal in the differential diagnosis. If there is an obstruction, a dilated mottled pylorus and widened canal, it is strongly suggestive of a prolapsed gastric mucosa.

Prolapse of the gastric mucosa is ordinarily a medical problem and in only the severe intractable cases with pain, and those with complications, such as hemorrhage and obstruction, is surgery indicated. One of the principle factors to overcome is the accelerated peristaltic activity. The use of antispasmodics, anticholinergic and ganglionic blocking agents offers the ideal means to control or eliminate this phenomenon. The administration of these drugs may be useful as (1) a physiologic test, and (2) a therapeutic test for the relief of symptoms. The clinical improvement following the use of these drugs should be commensurate with the salutary effects observed clinically and roentgenologically. The use of antacids is usually unnecessary in the uncomplicated case, since the vast majority of cases show no evidence of hyperacidity. Owing to the frequency of the influence of psychic phenomena associated with this condition, it is well to employ psychiatric therapy in order to eliminate this phase of stomach activity.

A number of different operations have been recommended for the cure of prolapse of the gastric mucosa, namely, (1) simple excision of the redundant mucosa, (2) excision of the mucosa with pyloroplasty, (3) pyloroplasty, (4) gastric resection, and (5) gastroenterostomy. Subtotal gastric resection is the

operation of choice for the total eradication of this condition. In 181 collected surgical cases, the type of operation was mentioned in 103. A simple excision of the redundant mucosa was done in 9, an excision with pyloroplasty in 34, pyloroplasty in 4, gastric resection in 51, gastroenterostomy in 4, and a gastrostomy in 1 case. It is important to point out that all of the operated cases were not cured of their symptoms. It is highly probable that in many instances the prolapse is an incidental finding which is not related to the patient's clinical problems. Rappaport et al, Castren, and others have directed attention to the poor results encountered in some instances following operative measures.

Summary

Further studies on prolapse of the gastric mucosa is presented with additional pertinent data on this subject. Up to 1954, there have been 146 published reports on prolapse of the gastric mucosa, with a total of 1,066 cases recorded. Among these, there were 209 cases explored surgically. The importance of this condition is emphasized as a clinical entity and a source of symptoms resembling an atypical duodenal ulcer. The characteristic differences between the symptoms of prolapse of the gastric mucosa and duodenal ulcer are enumerated. The diagnosis is best made by means of the roentgen examination. The roentgenologic findings are pathognomonic of this condition. The incidence is approximately 10 per cent in the general adult population. The condition is usually a medical problem. Only in the severe or intractable cases associated with complications is surgery indicated. In the medical treatment, antispasmodics, anticholinergic agents, diet, and psychiatric therapy are the necessary therapeutic measures. In the surgical treatment, when indicated, subtotal gastrectomy is the treatment of choice in most instances.

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CONGENITAL ANOMALIES OF THE DUODENUM

Report of Eleven Cases Representing as many Patterns

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The duodenum is a favorite site for congenital anomalies. These are confined to the 2nd, 3rd and 4th portions, which develop from the midgut anlage. The first portion, originating from the foregut, usually does not participate in developmental anomalies.

Fig. 1—Case 1. Huge redundancy of the 2nd, 3rd and 4th portions of the duodenum. The bulb is not visible. The 2nd portion, after an initial upstroke descends deeply, in a redundant, stepladder shape, left and behind the antrum. All the segments run in a three dimensional, wave-like curve, deep below the greater curvature.

The congenital malformations may be classified into two main groups. The first one comprises those serious, luckily extremely rare cases which are characterized by severe structural changes, foremost among them are the atresias. The duodenal atresia, according to Tandler, may represent the normal developmental state of the early intrauterine life of a second month embryo. Postnatal persistence of the epithelial occlusion, by failure of later opening of the obliterated lumen may explain the occurrence of the duodenal atresia after birth.

The second, a much larger group comprises those, usually interesting cases of duodenal malformations, which by their harmless nature rarely cause clinical symptoms or suffering to the bearer. For this reason they usually remain undiagnosed, or are only incidentally recognized. Redundancy, inversion and malrotation are the principal subgroups.

The first group, manifested by structural congenital anomalies, often interfering with normal functions, if not incompatible with life itself, will not be the subject of this paper. Our observations and remarks refer exclusively to the second group as classified in the above paragraph.

Characterization of the Congenital Anomalies

In congenital anomalies positional changes of the duodenal curve or in some of its segments predominate. These result from developmental arrests or im-

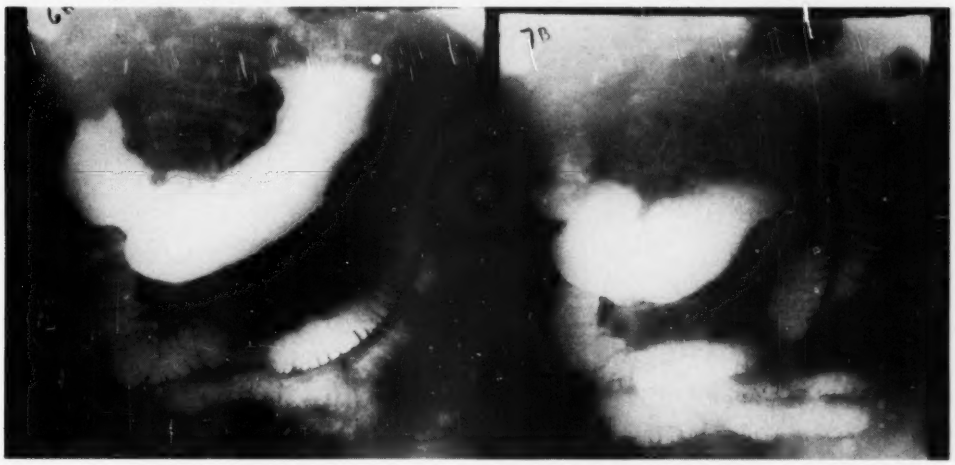

Fig. 2—Case 2. Redundancy of the duodenum. The 2nd, 3rd and 4th portions run in a huge arch, parallel with and considerably below the greater curvature. Note the wave-like undulations especially on film at left. Left and right films show slightly different configurations.

perfections of rotation, descent and attachment, as they succeed each other in the course of embryonic life. Genetic factors may be responsible for the developmental anomalies, complete or partial. They are found alone as individual observations, or in association with other developmental malformations.

These congenital anomalies are usually symptomless. Their recognition is often incidental and may depend upon the examiner's skill and experience. They can best be demonstrated by x-ray which surpasses anything that surgery or postmortem examination could offer. Most of the cases, however, remain, even if roentgenologically demonstrated, undiagnosed, because the changes and their significance may not be familiar to the physician or the radiologist, and thus escape attention. Only careful and well planned study will result in the detection, diagnosis and correct interpretation of these congenital changes.

Occasionally, these may prove to be of greater academic than practical significance. If they do not produce functional disturbances, affecting principally the motor power, as they usually do not, no symptomatology can be expected.

Classification

Various classifications are in use. The principal groupings of reduandancy, inversion and malrotation are apparently fairly well accepted. Sometimes the inversion is classified as a subgroup under malrotation. The inversion itself may embrace a variety of changes in the course of the individual duodenal segments. Their common denominator is that the second portion instead of descending

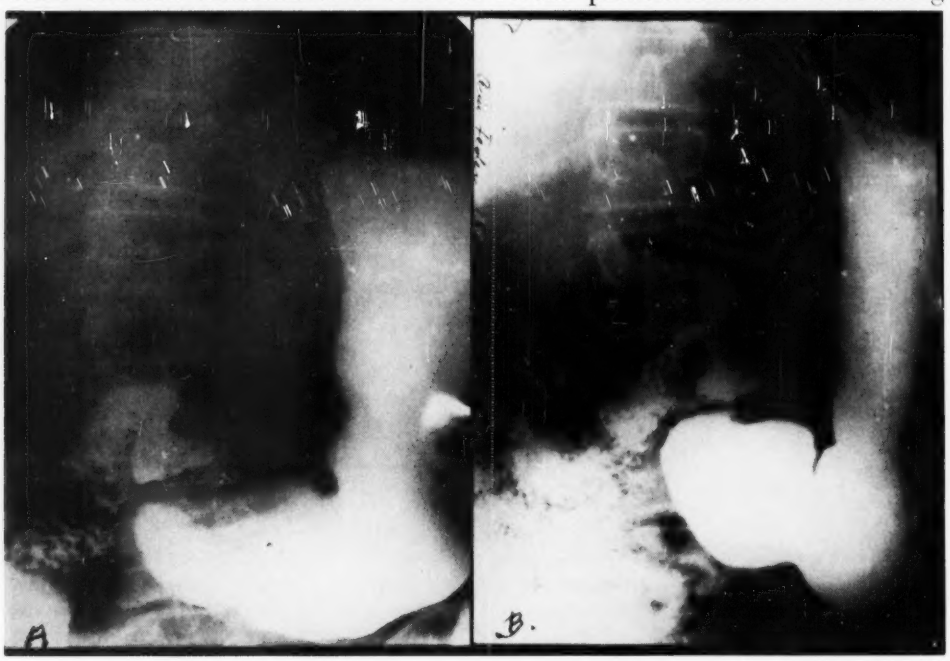

Fig. 3—Case 2. Both films show the abnormal duodenal curve, typical in malformation. The 3rd portion turns outward, toward the right, instead of to the left, inward.

shows an ascending course. Redundancy usually affects the proximal segment. Malrotation of the duodenum may be associated with malrotation of the jejunum, or even with the malrotation of the entire midgut anlage.

The congenital anomalies in the individual cases may produce changes affecting the length, course, curve, position and movability of any of the duodenal segments.

Rarely are two cases entirely similar. Feldman enumerates and radiologically demonstrates the individual subgroups. Buckstein is less enthusiastic about the absolute and rigid classification of each and every case, because as he states: "almost every anomalous variation in the course of the duodenal curve has

been described". Furthermore he emphasized, that: "these variations in the configuration of the duodenal curve ordinarily appear to have no clinical significance". My own observations, in full agreement with both statements, may be even more emphatic to the effect that aside from clear-cut instances there are many inbetween or transitional designs, especially among the inverted types, or in which various anomalies are found in combination. Such instances are e.g. inversion with mobile duodenum, or inversion with partial malrotation, or redundancy with either of the two other anomalies, or even the combined

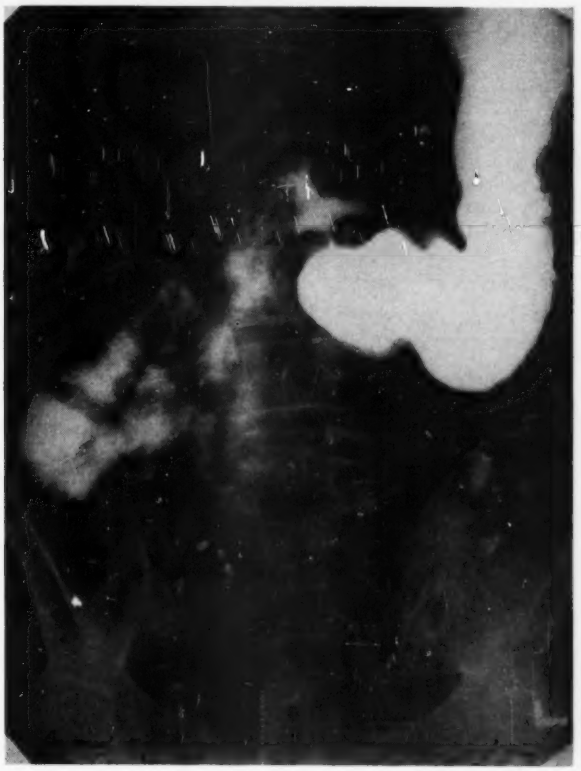

Fig. 4—Case 2. Malrotation of the duodenum and jejunum, in the presence of bulbar deformity, isolated visualization of the appendix, colonic diverticulosis and double gallbladder.

occurrence of inversion, reduandancy, partial malrotation and mobile duodenum with mesoduodenum. Classification in such cases is difficult or arbitrary. Nearly every case is of individualistic nature.

Eleven cases of congenital duodenal anomalies are presented here, all of them showing different configurations and patterns.

Report of Cases

Case 1:—R. G., married, white, female, 22, was first seen December 13, 1938. Emotional instability, nervousness, restlessness. Moderate obesity. B.M. minus

12 per cent. Blood pressure at times as low as 86/60. Severe constipation. Occasionally recurring sharp epigastric pain, especially after excitement or dietary excesses, severe enough to double her up. Alkalis of no avail. Excessive indulgence in smoking and consumption of coffee (10 cups daily).

Gallbladder x-ray negative. The appendix shows angulation. The distal third was visualized, but not the proximal segment. The duodenum showed *a huge redundancy of its 2nd, 3rd and 4th portions.* Starting from the first segment of normal appearance, the descending portion started off behind the antrum and its course was to the left instead of to the right of the bulbus. In one of the films redundancy was visualized in a step-ladder shape. The 3rd and 4th portions were gently undulating and generally ran in a huge arc before turning

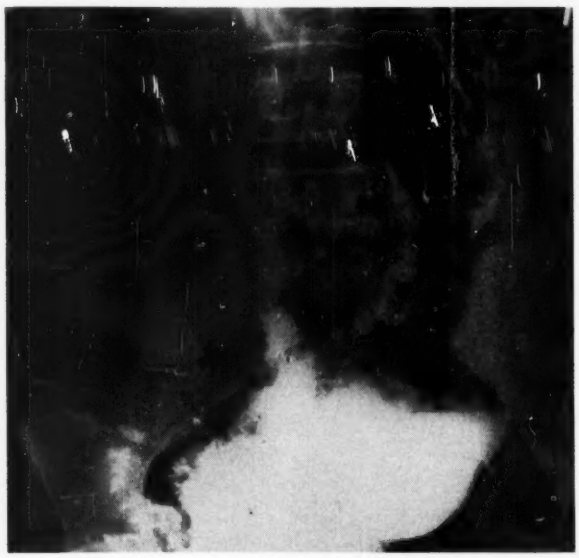

Fig. 5—Case 3. Abnormal duodenal pattern, of the inversion type. The duodenal curve was an irregular trigonum on a higher level. The descending portion took the course upward and to the left, the 3rd portion descended left to the vertebra.

towards the upper part of the fundus. Its course was parallel to the major curvature, considerably below this structure. Passage, emptying, markings and width of this organ were normal. Redundancy of the duodenum was an incidental finding. None of the complaints seemed to have had anything to do with the congenital anomaly, which, in itself, failed to produce symptoms.

Case 2:—A. F., white, married, female, 41, developed recurring epigastric pain at rare intervals during the last 5 years. X-ray revealed a bulbar deformity. There were associated anomalies, among them: *malrotation of the duodenum and jejunum.* Both were located in the right upper quadrant. The third portion of the duodenum instead of turning to the left turned to the right and joined the jejunum without angulation. The appendix, which showed a persistent isolated

visualization, was in the right lower quadrant, classifying the malrotation as an incomplete one. There was an extensive colonic diverticulosis. The colon was ptotic and redundant. The gallbladder was bilocular. Malrotation of the duodenum proved to be an incidental finding in association with several other congenital developmental anomalies and with some acquired pathologies. The duodenal malformation caused no clinical symptoms.

Case 3:—N. W., white, married, female, 49. She was referred for diagnosis on July 16, 1954. Her weight had dropped from 150 to 92 lbs. General ptosis and atony of the abdominal organs. The stomach was ptosed to such an extent

Fig. 6—Case 4. The superior segment is not visualized. The three other segments run in an upward concave curve, encircling the vertebra from three sides, grossly parallel to, and at a higher level than the lesser curvature.

that an 11 x 14 film was required for its enclosure. Mastoid operation 15 years ago. Pain and vomiting developed three years ago and a duodenal ulcer was diagnosed which cleared up. Recently arthritis of the spine was diagnosed. She underwent surgery for some injury of the shoulder. Exhaustion, nervousness, anorexia, weight-loss, insomnia, morning-tiredness, constipation and miserable feeling after meals were among her complaints. She was absent from her employment half of the time because of ill health.

Her blood pressure was 80/70. She was bony, skinny, dehydrated, exhibiting signs of Simmonds' disease. The stomach was ptotic and atonic. The cecum was hyperdescendent and the colon ptotic. The duodenum showed an unusual

congenital anomaly. It belonged to the *inversion* type. The cap was normal. The second portion, instead of descending, turned upward and to the left, crossing the 5th and 4th lumbar vertebrae, at a high level, obliquely, and after a sharp angulation from the left of the vertebra it turned downward and to the right, toward the bulbus, forming an arch. The duodenal curve described a trigonum, way above the pyloric level. Its axis was ascending toward the left, instead of downward toward the right.

Case 4:—M. D., white, married, female, 26. First seen July 2, 1937. She felt nervous, but suppressed her feelings. She tired easily and felt weak. Frequent colds, occasional loose bowels, headaches, dizziness, insomnia, weight-loss, stiff and painful sacroiliacs. Chronic indigestion. Sticking epigastric pain; queer complaints; achylia gastrica.

Fig. 7—Case 5. Abnormal duodenal pattern. The superior portion is elongated. The duodenal curve describes a huge circle.

Gallbladder x-ray was negative. The appendix revealed a persistent isolated visualization. The duodenum showed a congenital anomaly. The superior segment was not visualized. The three other portions ran in a curve with its *concavity pointing upward,* encircling the vertebra from three sides. The course was parallel to the lesser curvature and at a *higher level.*

Case 5:—A. R., married, white, male, 59. Referred for diagnosis on May 3, 1953. He was a known hypertensive; suffered from dizzy spells, hot flushes and recently developed retrosternal pain. Anxiety neurosis. Constant fear and worry concerning his health. Tired feeling; nervousness. Blood pressure 202/102 mm. Hg.; E.K.G. negative. X-rays taken 10 days after barium meal revealed isolated

visualization of the vermiform appendix (appendicular stasis) and very extensive colonic diverticulosis. Hundreds of symptomless diverticula were visualized in the distal half of the colon. The duodenum showed an abnormal pattern. The

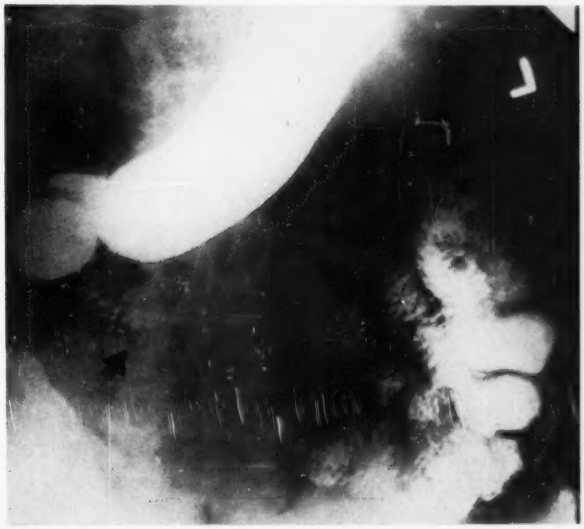

Fig. 8–Case 6. Abnormal duodenal pattern. The duodenum describes one huge curve, without demarcation of its individual segments, below the greater curvature. Note the 2 jejunal diverticula (arrows).

first portion was elongated, representing *redundancy,* the arc ran in a *small circle,* which, considering a body weight of 225 lbs., and a height of well over 6 ft., was relatively of a shorter size.

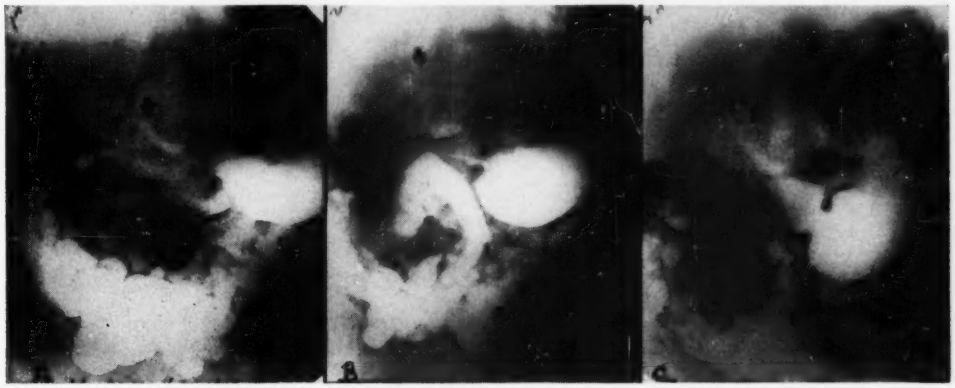

Fig. 9–Case 7. Unusual duodenal pattern. The superior segment is elongated, it points upward and toward the right; the descending portion is very short. The third segment runs at a high level; the fourth portion is abrupt and tall. The duodenal curve can be most clearly seen on film C. Follow the arrows.

Case 6:–A. H., white, married, male of 72. Referred for diagnosis on September 5, 1947. Dizzy spells. Numbness in extremities. Limited motion. Systolic blood pressure registered over 230 mm. Hg. reading. Developed severe retro-

sternal pain a week previously accompanied by respiratory difficulties. Experienced no gastric complaints other than heaviness after meals which was relieved by eating small amounts at frequent intervals. Gas-distress and constipation. X-ray examination revealed *two huge diverticula in the jejunum. The duodenum ran in a huge curve,* parallel and below the major curvature. The segments and their delineation into each other remained unmarked. The course of the descending portion was to the left of the pylorus instead of to the right. The duodenojejunal junction was completely missed.

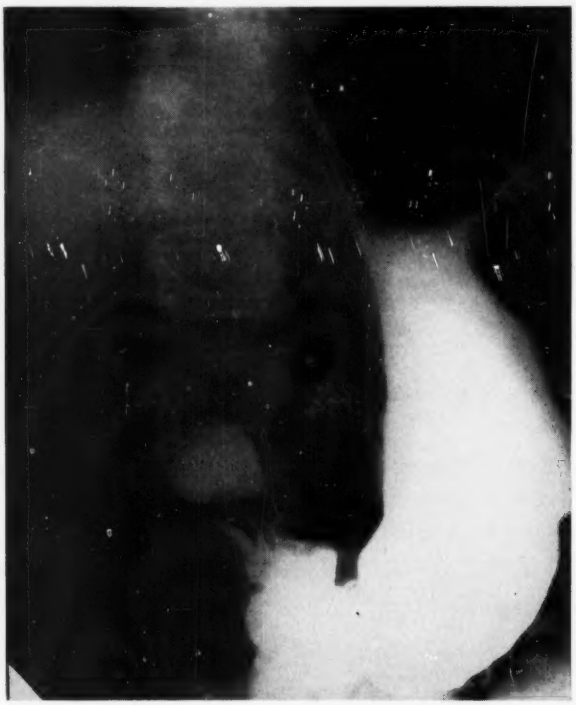

Fig. 10—Case 8. The duodenal bulb is normal, it points upward; the descending segment is very short and runs at an angle toward the right; the 3rd portion crosses the vertebra at the level of the pylorus. The duodenal curve is shortened and runs at a high level.

Case 7:—O. N., widow of 70. Anxiety neurosis. Attacks of auricular paroxysmal tachycardia, occurring at rare intervals, sometimes lasting for several days. Blood pressure 175/80. E.K.G. revealed a T-4 inversion and occasional premature ventricular contractions. There was a huge redundancy of the splenic flexure. The duodenum showed an abnormal pattern. Its first portion was elongated, somewhat *redundant* and ascending, the descending portion was *very short,* thereby the third and fourth portions ran at an *abnormally high plane;* its jejunal junction formed a sharp angle.

Case 8:—E. S., white single woman, 50. First seen July 2, 1944. Psychoneurotic patient, with her complaints concentrated around the gastrointestinal

tract. Most important among these were her gastric complaints of 15 years duration and certain severe painful burning sensation at the appendicular area of six months' duration. Gas-distress, gaseous eructation, anorexia, fullness, con-

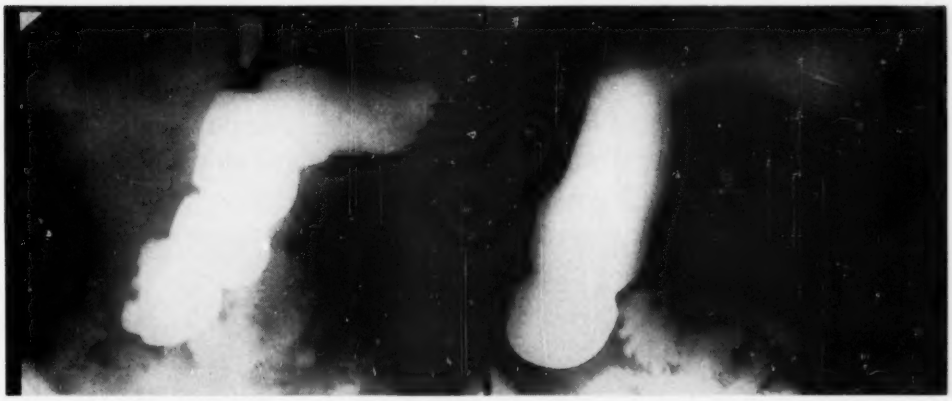

Fig. 11

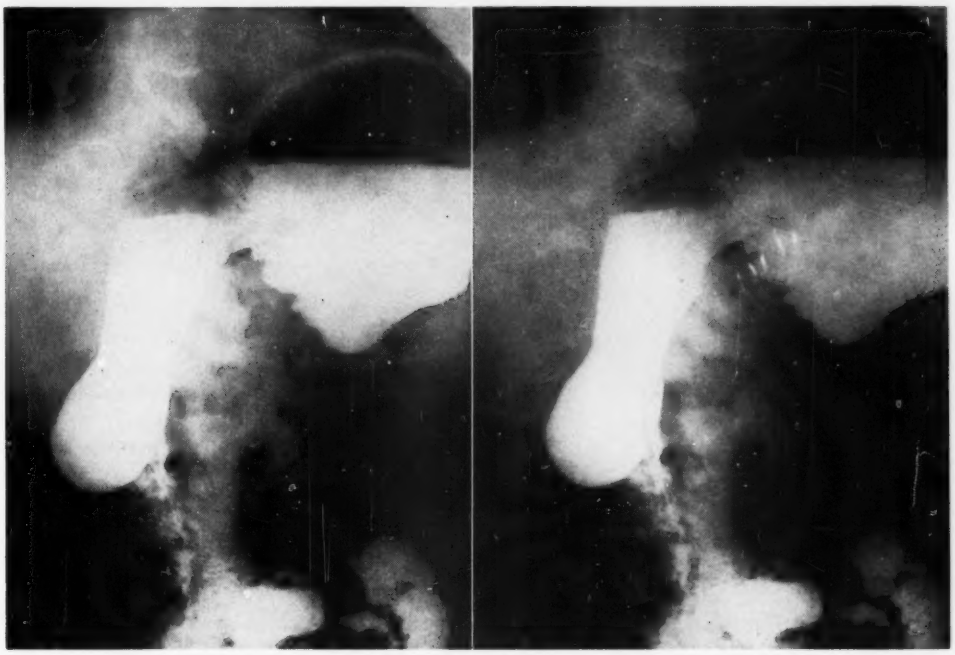

Fig. 12

Figs. 11 and 12—Case 9. Duodenal pattern in cascade stomach. Both films on both figures show various fillings of both chambers, at various times. The upper loculus is by far larger, if filled, it weighs heavily and hangs down.
The duodenum, located to the left of the antrum, runs downward and to the left, without any demarcation line.

stipation were her additional symptoms. She claimed that whereas food intake had no effect on pain or burning, milk or water intake increased them both.

She was anemic; lost no weight. During the day she was somnolent; at night insomniac. Symptoms of neurasthenia. Marked tremor on fingertips and eyelids. E.K.G. was negative. On exercise tolerance test development of auricular and ventricular premature beats. On psychic approach and conservative medical treatment patient's condition improved and she lost all her complaints. Her remaining query was: "where did all the pain and burning that I had, go to?"

X-ray of the gallbladder was negative, as was the gastrointestinal tract, with the exception of the duodenum, which showed a congenital anomaly. The duodenal bulb was normal, pointing upward. The descending segment was very short

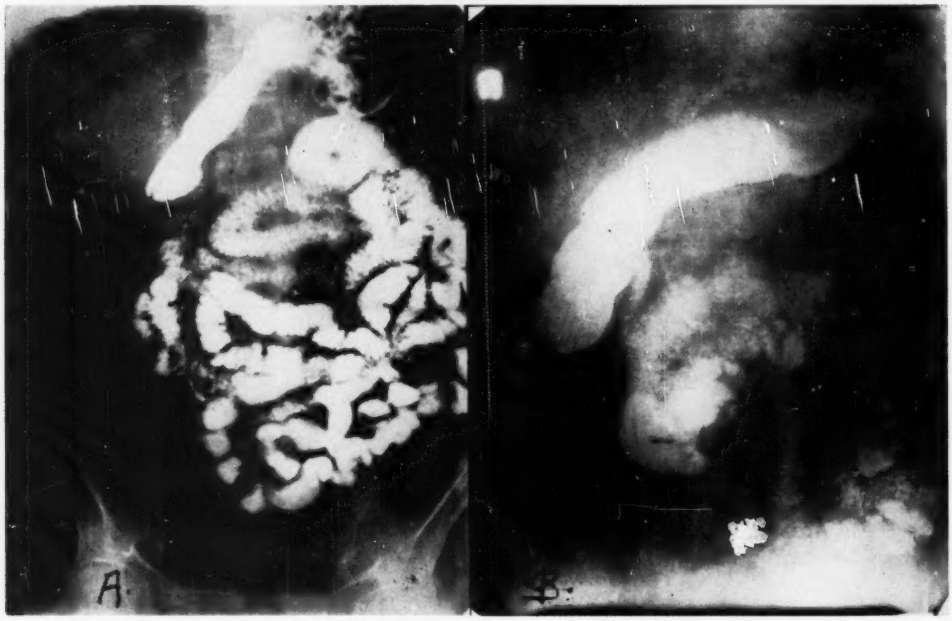

Fig. 13—Case 10. Duodenal pattern in case of inverted stomach. It runs medially from the antrum. Delineations of its segments are missed. Abnormal shape and position.

and ran in an angle toward the right. The third portion crossed the vertebra at the level of the pylorus. The *duodenal curve was considered shortened, running at a high level.*

The case contained elements of *iatrogenic origin.* She was treated by her previous physicians for such diagnoses as "gallbladder-trouble", "gas", "fallen stomach", and "ulcers", etc., in which she was disappointed. Failure of results apparently aggravated her condition and kept her is a state of excitement and anxiety. She felt frustrated.

Case 9:—M. K., married, female, white, 46, first examined October 15, 1935. She was treated for some gallbladder condition for many years. During the past 5 to 6 years she complained of gastric cramps which radiated to her back. Pain

was not severe enough to double her up. Its type was hunger-pain with postprandial improvement. Her nights were free of pain. Each painful period lasted for about 2 or 3 weeks. It recurred in cycles. Pain, regardless of food intake or quality returned typically three times a day. Characteristically heavy food was better tolerated. Frequent nausea and vomiting. The vomited masses contained 4 or 5 ounces of bitter liquids. Constipation. Nervousness; attributed to the gastric ailment.

Scoliosis. "Hernia lineae albae" with a foramen of a palm's size. Three children, all delivered by Caesarean section.

Radiography revealed a cascade stomach of huge dimensions. The major curvature at its upper third was deeply retracted. This retraction divided the

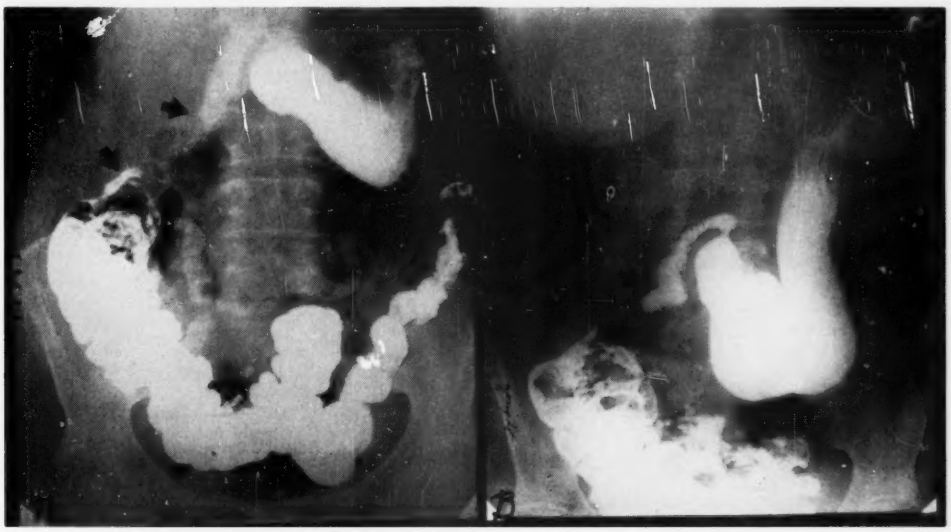

Fig. 14—Case 11. Both films show the descending portion of the duodenum pointing toward the right, instead of the left. On film A the lower arrow points toward the appendix, a subhepatic localization due to inversion of the cecum. The transverse colon is drooping and redundant.

stomach into two chambers. The upper loculus was huge and when filled, hung down by its gravity. This filled up to the size of an average stomach before it started to spill over into the lower chamber. Gas accumulation in the splenic flexure.

The duodenal bulb was invisible, behind the antrum. The *duodenal segments ran downward and toward the left* without demarcation lines and without the delineation of the duodenojejunal junction. There was no resemblance to any normal anatomy. This pattern conformed to the picture of a cascade stomach, whether of primary or of secondary development, and whether congenital or acquired. The former represents *true malformation, whereas the* latter is only a *malposition.*

Case 10:—M. W., white, married, female, 50. This highly neurotic, oversensitive and overactive patient exhibited a series of congenital anomalies. Huge redundancy of the splenic flexure, interchangeably shifting to sigmoidal redundancy, hypodescent of cecum, inversion of the stomach with duodenal anomaly were the gastrointestinal changes. There was a diverticulum of the gallbladder. The duodenum descended *downward and toward the left* without demarcation of its portions and of its junction with the jejunum, rather similarly to the previous case of a cascade stomach. In this case the anomalous duodenum with a congenitally inverted stomach represented parts of a series of congenital malformational anomalies.

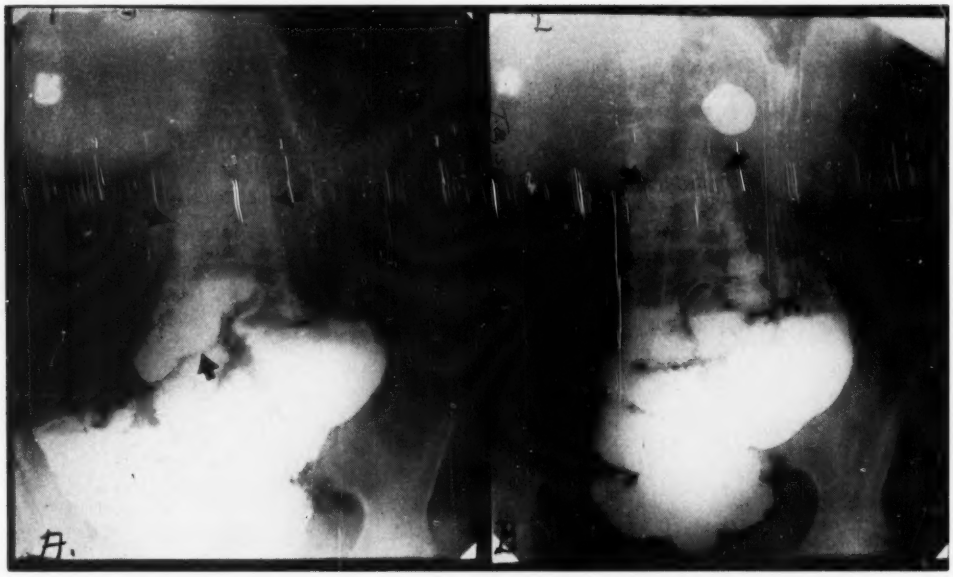

Fig. 15—Case 11. In upright position the duodenal curve describes a trigonum, its sharp angle pointing upward. Its upper point approaches the cardia. The bulb is not visualized, the descending segment points downward and to the right; the third portion shoots high up and forms a sharp angle with the fourth segment, which runs vertically downward, left to the vertebra.

Case 11:—T. S., married, white, female, 39, first seen July 23, 1940. An exceedingly nervous woman, who exhibited among several congenital anomalies such as an inverted cecum, subhepatic appendix, ptotic and atonic transverse colon, also an unusual anomaly of the duodenum. Diagnosis had been made in 1940 and reaffirmed in 1953. The duodenal bulb was never completely filled and visualized. The second portion, after an initial descent turned, as a sign of malrotation, outward instead of inward, and then exhibited a huge inversion with the segment running upward, opposite in direction to the normal, and reaching a height, at the approach of the cardiac region of the stomach. At this point, in the concavity of the lesser curvature, the duodenal segment turned sharply downward to complete a triangle with a narrow base. Here, *malrotation*,

inversion, redundancy and extreme mobility ("duodenum mobile"), and *a mesoduodenum* of the 3rd and 4th portions were diagnosed. This was the observation on films taken in upright position. On films taken *in prone* position the inversion and high ascent of the malrotated segment *disappeared.* The duodenal curve revealed a practically *normal pattern.* The huge and free movability of the duodenal segments and a chage of their patterns in prone position is explainable only on the assumption of a huge and free mesentery, i.e. a mesoduodenum, a congenital anomaly, in itself. The findings in 1940 and in 1953 were identical.

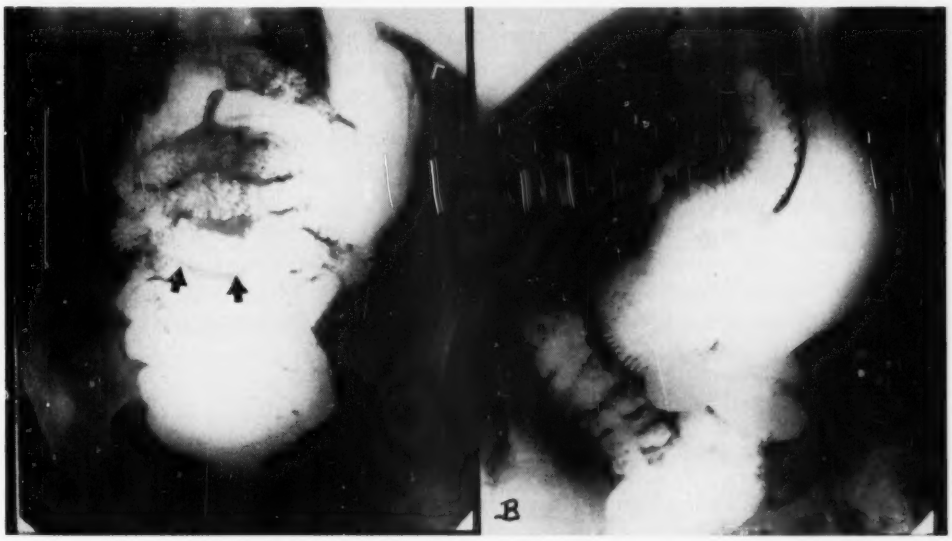

Fig. 16—Case 11. A. Arrows point to the course of the duodenum. Prone position. The duodenal curve is below the greater curvature.
B. Upright position. The duodenal curve shoots way up above the lesser curvature.

Comments

Eleven cases of congenital anomalies of the duodenum were presented. Some fitted into one of the accepted groups, others almost required their own classifications. Nearly every case appeared in its own individual design. Because the variations in several patterns produced bizarre and not easily classifiable shapes and configurations, their rigid groupings into one of the known classes of redundancy, inversion and malrotation might easily have proven factitious or arbitrary.

All these congenital anomalies were incidentally detected during routine x-ray examination of the gastrointestinal tract. No symptoms were present and no pathology at this particular site was expected. In nearly all the cases additional congenital anomalies were detected either in the gastrointestinal tract, or in the gallbladder. Structural pathology or some borderline change, such as an

appendicular stasis, or diverticulosis of some segment in the gastrointestinal tract were relatively frequent.

None of these anomalies can be considered as a disease entity. They rather represent stigmata, or express genetic errors, often in constitutionally inadequate individuals. Consequently, due to these anomalies, no symptomatology, or clinical course is to be expected. The prognosis is "per se" good and no treatment is required.

Summary

Eleven cases of congenital duodenal anomalies appeared in somewhat different shape, configuration and position. Practically every case exhibited a design of its own.

None of the cases had signs or symptoms referable to this anomaly or to any specific pathology. Their detection was incidental. Association with other congenital anomalies was frequent. Diagnosis was made by x-ray demonstration of the abnormality.

These anomalies are rare. Their recognition may be missed, because even if they are demonstrated by x-ray, they are not necessarily recognized, diagnosed or correctly interpreted.

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CHOLECYSTOGASTROSTOMY OF 28 YEARS' DURATION*

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and

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We are presenting a case of cholecystogastrostomy of 28 years' duration. The literature carries only scant reference to longevity or compatibility with good health over an extended period of years. As far as we could ascertain, the longest follow-up cases mentioned in the literature have been approximately five years.

The operation of cholecystogastrostomy was utilized in the past for the purpose of circumventing irreparable obstruction to the common duct such as carcinoma of the head of the pancreas, stenosis of the common duct, congenital deformity or obliteration of the duct and enlarged glands at the hilum of the liver. Its use was also extended as a substitute for gastroenterostomy in gastric ulcer. Babcock[1] and Frenkel[2] recommended the anastomosis of the gallbladder directly into the ulcerated area in cases of chronic gastric ulcer which did not respond to medical treatment. The assumption was that the bile would neutralize the gastric acidity.

Murdy[3] suggested that the operation be used in place of cholecystostomy for infection of the gallbladder without stones or for cholangitis with or without stones. Others advised this procedure for long continued drainage in infective biliary cirrhosis associated with jaundice. Cholecystogastrostomy was considered superior to cholecystostomy in that better nutrition was maintained. DuBose[4], Deaver[5], Braithwaite[6], Villard and Richer[7] urged wider use of this operation in the surgical management of perforated gastric and duodenal ulcers, perforation of the gallbladder and obscure chronic or intermittent jaundice.

During the course of years, cholecystogastrostomy was looked upon less favorably because clinical and animal experimentation seemed to indicate that this operation resulted in infection of the gallbladder and abscesses throughout the liver. The cause of these ascending infections was ascribed to the fact that the biliary system was no longer protected by the sphincter of Oddi and consequently a reflux of contents from the stomach to the gallbladder occurred. Various surgical modifications of the anastomotic site were devised in attempts to overcome this obstacle. McCaughan and Purcell[8] described what they considered a satisfactory method of preventing the reflux of bile by a simple type of valvular cholecystogastrostomy.

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In reviewing statistical reports, Whipple[9] ascribed the frequent occurrence of ascending infection with cholangitis as the probable cause of the short life expectancy in cases of cholecystogastrostomy. On the other hand, our case may well indicate that a fistulous tract between the gallbladder and stomach is not as deleterious or hazardous as previously suspected.

Case Report

D. R., a 47 year-old male, was referred to the gastrointestinal clinic after having been treated in the medical and neurological clinics in June, 1951.

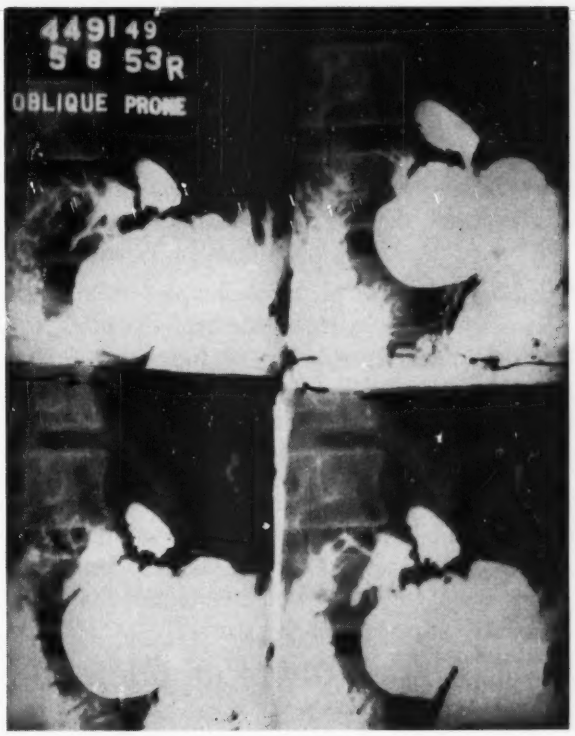
Fig. 1

The patient's chief complaints were that of pain in the epigastrium or right upper quadrant, bloated feeling, belching and occasional self-induced vomiting.

His past history is interesting. In 1926, he developed a severe jaundice for which he was hospitalized at the Post Graduate Hospital. At operation it was found that the liver was about twice the size for the patient's body weight and age. The gallbladder was thickened, without calculi. It did not contain bile but a mucoid fluid suggesting edema or obstruction of the cystic duct. The

common duct was narrow and seemed thickened. A cholecystogastrostomy was performed, the point of application being in the lesser curvature, 3 cm. from the pylorus. He made an uneventful postoperative recovery and the jaundice cleared.

In 1945 he was admitted to the Jewish Hospital of Brooklyn because of complaints of epigastric pain and general abdominal discomfort. The pain was spasmodic, came on suddenly and lasted for hours or days. They were associated with frequent heartburn, belching and occasional nausea and vomiting. The pains were temporarily relieved by belladonna and phenobarbital.



A B

Fig. 2

During his hospitalization the pertinent laboratory data was as follows: Blood sugar–110 mg.; blood urea–18 mg.; B.M.R.– –20; Urinalysis–Negative for sugar and albumin; Hgb.–91 per cent; R.B.C.–4,610,000; W.B.C.–9,900; Polys–69; Lymphs–29; Monos–2.

Oral cholecystography with Priodax failed to satisfactorily visualize the biliary sac. No calculi were noted.

The x-ray studies of the gastrointestinal tract were interpreted as revealing a small pocket at the pyloric site, suggestive of a pyloric ulcer.

The patient was discharged March 16, 1945 with a diagnosis of 1. Poor biliary function. 2. Pyloric ulcer.

At present the patient's chief complaints are epigastric pain, discomfort in the right upper quadrant with occasional radiation to the back. The pain subsides at midnight. He has no heartburn but belches a good deal. His appetite is good, bowel movements regular, no melena, weight is stationary.

Personal history:—The patient readily admits to being a psychological problem with severe tension and anxiety states. He has been involved in extremely disturbing marital and sociological problems for many years.

Physical examination revealed a thin, middle-aged man, voluble, tense and anxious. The pupils react to light and accommodation, fair hemic component. The heart and lungs were within normal limits. Examination of the abdomen revealed a long, well-healed right rectus scar extending from the epigastrium to the lower abdomen. The liver and spleen were not palpable. There was a moderate gallbladder tenderness and slight tenderness to liver percussion. No palpable masses.

Laboratory findings:—Gastric analysis—samples clear with a slight yellow tinge. Fasting specimens:—Free hydrochloric acid—28 units; total acid—62 units; Bile—Positive. Thirty minutes after histamine injection:—Free hydrochloric acid —62 units; total acid—108 units; Bile—Positive. Serum Amylase—61; Serum Lipase—1-4; Thymol Turbidity—2.1; Total Cholesterol—197 mg. per cent; Free Cholesterol—55 mg. per cent. Stool examination—Negative for parasites or ova.

Gastrointestinal Series:—December 21, 1951 (Fig. 1). Radiographic findings revealed the following: The barium can be seen passing into the biliary radicals. The duodenal cap is irregular suggesting a duodenal ulcer. Five and twenty-four hour films show normal emptying of the stomach. No free gas can be seen within the biliary radicals throughout the study.

Oral cholecystography fails to visualize the gallbladder.

The patient was treated in the gastrointestinal clinic up to September 15, 1952. Therapy consisted of a bland diet, Banthine, Syntropan, mild sedation and reassurance. During the course of follow-up, it became apparent that the patient was taking codeine. He was at the same time getting psychiatric guidance. He declined any gastroscopic examination.

Upon request the patient returned for a gastrointestinal x-ray study on May 8, 1953 (Fig. 2). The duodenal cap was persistently irregular and deformed with a suggestion of a niche in the lower mesial portion. Adjacent to the duodenal cap may be seen a small, fistulous tract (Fig. 2B) and a large pocket of barium, superiorly, indicative of a still patent cholecystogastrostomy.

The patient at present is employed, feels comparatively well and is more or less resigned to minor dyspeptic symptoms.

Summary and Conclusions

This case is presented because of many interesting aspects. A patient having a cholecystogastrostomy for 28 years is in itself most unusual. He has had no recurrent jaundice since the operation. The patient's laboratory tests show no indication of cirrhosis. Throughout the postoperative years there has been no evidence of either ascending infection or hepatitis. He still has a high gastric acidity. It is very likely that the presence of a high acidity has been instrumental in preventing ascending infection. One may speculate that later in life if he were to develop an achlorhydria there may be the possibility of ascending infection.

The patient developed a duodenal ulcer contrary to the previous belief that the presence of bile in the stomach would prevent ulcus formation by neutralization of the acid.

The fact that in oral cholecystography the gallbladder was not visualized is not unusual in cholecystogastrostomy. The dye passes rapidly through the gallbladder without being concentrated.

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SKIN AND ORAL LESIONS, ATTRIBUTABLE TO NUTRITIONAL DEFICIENCY, ASSOCIATED WITH CIRRHOSIS OF THE LIVER

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Search of the literature has revealed that little or nothing has been published on the subject of cutaneous lesions of nutritional origin associated with liver cirrhosis. After noticing this type of lesions in individuals suffering from atrophic cirrhosis of the liver we became interested in the subject and recorded the dermatoses encountered in patients admitted to our clinical service.

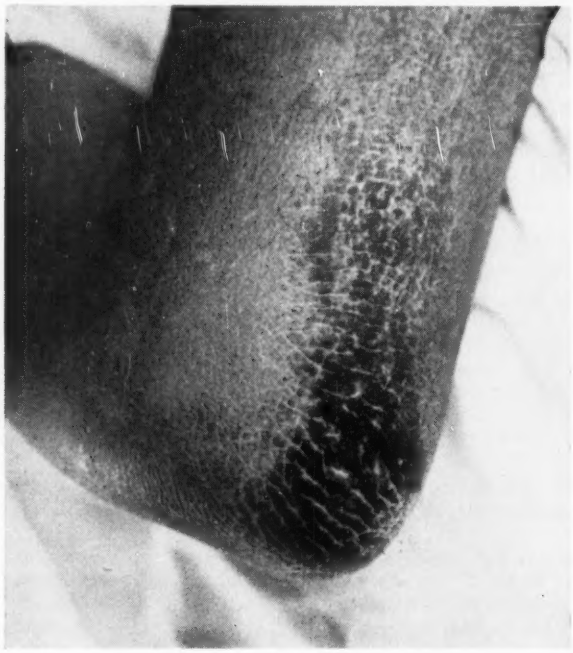

Fig. 1—Hyperkeratotic and hyperpigmented lesions at the elbow of a cirrhotic patient.

A group of well-studied cases were presented[1] at the Fifth Brazilian Congress of Gastroenterology but now, with information from additional cases, we are able to discuss the subject with more confidence.

Sixty patients with liver cirrhosis have been observed, 40 male and 20 female, the youngest being 15 years of age and the oldest 65.

Almost all of the patients had lesions of the nutritional type on the skin and oral mucosa: 49 had cutaneous lesions and 48 had buccal lesions.

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In addition to the cases of liver cirrhosis, 60 other patients, without cirrhosis, were also examined. These patients were from the same wards and had essentially the same standard of living as the cirrhotic patients. Only a small proportion of the noncirrhotic patients had skin or oral lesions of nutritional origin–(two with oral lesions and sixteen with skin lesions). Moreover, the lesions were much less severe than those of the cirrhotic patients.

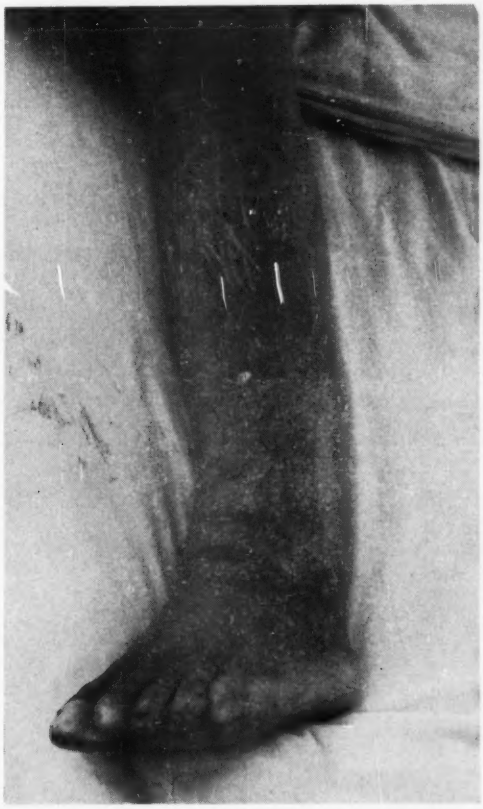

Fig. 2–Xerosis of the leg and hyperkeratosis of the cutaneous folds corresponding to the tibiotarsal articulation in one of our cirrhotic patients.

Oral Lesions

Glossitis was the most frequently encountered condition, being observed in 48 of the cases. Sometimes it was represented only by hypertrophy of the fungiform papillae with or without furrows of various depths. In other cases there was general atrophy of the papillae giving to the surface of the tongue a very smooth and bright appearance.

Cheilosis (2 cases) and angular stomatitis (one case) were rarely encountered. Mild labial and lingual blood suffusion was observed in one case.

Cutaneous Lesions

Of the cutaneous lesions, xerosis and hyperkeratosis with hyperpigmentation were the most commonly found.

The former was present in 46 cases, sometimes it was generalized but the preferential sites were the legs and the external aspects of the forearms.

The hyperkeratotic lesions had the following distribution:

Dorsal surface of the feet	31 times
Elbow	21 "
Legs	20 "
Malleolar region	7 "
Cutaneous folds corresponding to the tibio-tarsal articulation	7 "
Knees	3 "
External aspect of the thigh	1 "
Dorsal surface of the hands	1 "

Fig. 3–Typical "crazy paving" pattern of the skin of the legs of a cirrhotic patient.

Pellagroid dermatitis, subcutaneous blood suffusion on the dorsal surface of the hands and forearms, seborrhea and comedonnes in the nasal region were each encountered once.

Discussion

In the light of these observations one may conclude that oral and cutaneous lesions of nutritional origin occur frequently among cirrhotic patients in the ascitic phase. This association has not been reported in the literature notwithstanding its easy explanation since it is now widely believed that cirrhosis of

the liver is a primordially nutritional disease. Thus one may attribute both the hepatic and the cutaneous lesions to a common cause: nutritional deficiency.

On the other hand, the cirrhosis itself, implying a certain degree of hepatic dysfunction, without doubt causes serious interference with the metabolism of vitamins and other nutritive substances such as proteins. This can give rise to a state of severe malnutrition with possible manifestations on the skin and mucosa. Therefore, there are two possible mechanisms which might explain the etiopathogeny of the skin and oral lesions we have observed.

In the light of the lesions we described above one may conclude that the nutritional deficiencies of the patients were chiefly avitaminoses PP and A. Glossitis and hyperkeratosis with hyperpigmentation were the most frequently encountered and these, at least classically, are attributed to the lack of these two vitamins. It must be remembered, however, that in vitaminology there are

Fig. 4–Hypertrophy and pigmentation of fungiform papillae in a cirrhotic patient, giving to the tongue a stippled appearance.

still many facts to be clarified. Interpretations that were generally accepted yesterday are greatly modified today and those that seem absolutely correct today may be greatly changed tomorrow. For instance, glossitis, so frequently seen in the cilinical picture of pellagra and for that reason classically considered as dependent on a deficiency in the PP factor, is also encountered in ariboflavinosis, sprue, pernicious anemia, etc.

Afonsky[2] cured glossitis in his patients with the PP factor as well as with B_2, B_1 and the whole B-complex.

Kruse, Sydenstricker, Sebrell, and Clerckley[3] have observed, in individuals on a basal diet supplemented with thiamine, ascorbic acid, niacin, and cod-liver oil, glossitis curable only with riboflavin and recurring after administration of this vitamin was stopped.

Field, Green, and Wilkinson[4] have reported six cases of glossitis which were not cured with nicotinic acid and which were cured completely by the administration of calcium pantothenate.

Rosenblum and Jolliffe[5], in an individual on a diet containing adequate amounts of nicotinic acid, thiamine, ascorbic acid and Vitamin A, caused the disappearance of glossitis by using pyridoxine. These same authors reported other cases in which glossitis, not influenced by treatment with nicotinic acid and riboflavin, healed completely after administration of Vitamin B-complex.

Thus, one cannot say categorically to which vitamin deficiency glossitis is attributable. Evidence indicated that nyacin plays an important part but

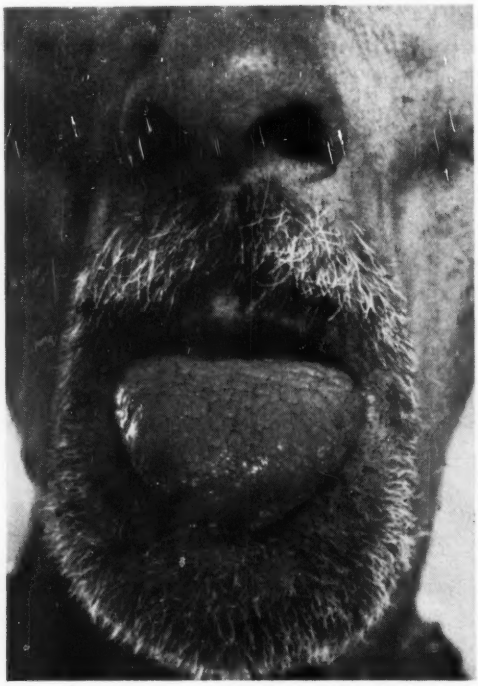

Fig. 5—Typical glossitis with deep furrows in one of our cirrhotic patients.

one must not overlook the part that synergism and antagonism of vitamins may play.

With reference to the type of skin lesions encountered in so many of our patients it may be said that most workers consider that they are attributable to deficiency of axerophthol.

In this connection it should be remembered that the cutaneous picture in so-called chronic pellagra may present lesions of the same kind. Since it is very difficult to identify an isolated vitamin deficiency and it is very common to

find what may be called pluricarencial syndromes, to which we called attention recently[6], the interpretation of the cutaneous signs in chronic pellagra may need revision. Perhaps certain aspects of the clinical picture in pellagra may depend on avitaminosis A or on a protein deficiency. Even the lesions encountered in our patients may be atributable to a protein deficiency the cutaneous manifestations of which are still unknown.

Although positive proof is lacking, we may say that the lesions encountered in our cases were probably attributable to multiple vitamin deficiency, and particularly to lack of nicotinic acid and Vitamin A.

Nevertheless, whatever the cause of the lesions, we believe that their frequent occurrence in patients with hepatic cirrhosis in the ascitic phase should be emphasized. They suggest that the patients are in an advanced state of malnutrition and indicate once more the usefulness of vitamin therapy in cases of liver cirrhosis.

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PSEUDOCYSTS OF THE PANCREAS

A Modified Method of Drainage Combined with Cystogastrostomy

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The diagnosis and treatment of cysts of the pancreas has been an interesting problem ever since they were first described by Morgagni in 1761. Cysts of the pancreas have been classified by Thigpen as:

1. Retention cysts. These are usually due to obstruction of the ducts by stones, and are true cysts, i.e. an epithelial lining is present.
2. Neoplastic, including cystadenoma, cystadenocarcinoma, teratoma.
3. Congenital, due to defective development. These are most common in infants, and may be associated with polycystic disease of the kidneys.
4. Pseudocysts, described below.
5. Parasitic. These are rare in the United States.
6. Miscellaneous, including hemangioma, endothelioma, cystic hemangioma, etc.

The most frequent type of cyst associated with the pancreas is the pseudocyst. As the name implies, it is not a true cyst, but a collection of material, usually liquid, surrounded by a sac of fibrous connective tissue, and without a lining epithelium. It is this absence of an epithelial lining that differentiates the pseudocyst from a true cyst. Since a true cyst lining may be incomplete or destroyed at times, only a pathological examination can differentiate the two conditions in some cases. The pseudocyst, as we shall see, is usually found in the peripancreatic tissue of the lesser omental sac, and only rarely within the pancreas itself.

Incidence

The pseudocyst is most common between the ages of 30 to 50 years, with about equal distribution between the sexes. In Brilhart and Priestley's series[1] of 44 cases from the Mayo Clinic 25 cases were in males and 19 in women. In a series reported by Meyer, Sheridan, and Murphy[2] the division was about equal in their 31 cases.

Pathological Physiology

The two causes of pseudocysts of the pancreas are (1) trauma, and (2) inflammation.

Trauma is the causative agent in from 5 per cent to 50 per cent of cases in various series reported. In the Brilhart and Priestley series only 2 of 44 cases gave a history of trauma. Schmeiden and Sebenig as quoted by Brilhart and Priestley found trauma significant in 16 of 128 cases collected by them, and Koerte found a history of trauma in 33 of 117 cases of pseudocysts. A history of trauma was present in 51 per cent of 27 cases reported by Meyer et al[2]. The effect of trauma to the pancreas is especially harmful since it must be remembered that the pancreas is immobile, and lies transversely across the abdomen against the spine. Furthermore, the pancreas is the only parenchymatous organ which reacts to trauma or inflammation by self-destruction. A sudden trauma, as a severe blow to the abdomen, may cause a hematoma of the organ with or without an inflammatory reaction. The rupture of the gland lining and acinar damage releases pancreatic ferments into the lesser omental sac causing a chemical peritonitis. This, plus the resulting inflammatory exudate, stimulates a fibrous connective tissue reaction, and, if the Foramen of Winslow is sealed, results in the encapsulation in a cyst of liberated ferments and exudate.

The most common cause of pseudocyst is a previous pancreatitis, acute or chronic. In Brilhart and Priestley's series of 44 cases, 21 cases had definite evidence of a previous pancreatitis, and in 21 more cases all the evidence pointed to an associated pancreatitis. As a result of the inflammatory process, calculi or many cysts may form, as in Edlin's case[8]. Rupture of the swollen acini releases the activated ferments, and we have the same succession of events previously described following trauma to the pancreas.

The size of the pseudocyst may vary from a few centimeters to 30 centimeters or more in diameter. The pancreas normally secretes 800 c.c. daily, and we may find a few c.c. to 3 liters of fluid in the cyst. Mahorner and Mattson[3] have found microscopically the contents of the cyst to contain epithelial cells, erythrocytes, leucocytes, fat, crystals of fatty acids, cholesterol, hematin, and necrotic tissue. The chemical contents include serum albumin, globulin, mucus, peptone, hemoglobin, leucine, tyrosine, and sugar. The enzymes found in the cyst are starch, fat, and protein-splitting. Meyer et al[2] found ferments in 9 of 12 cases specifically studied for enzymes. In the case described in this report amylase was present in the cyst fluid.

The pseudocyst is usually unilocular, in contrast to the cystadenoma or cystadenocarcinoma which are often multilocular. Maingot[13] found that 10 per cent of pancreatic cysts are malignant.

The position of the cyst depends on its origin in the gland, with the cyst enlarging in the path of least resistance. It is most frequently presented through the gastrocolic omentum, and less often through the gastrohepatic omentum. The retrogastric position is as common as the gastrohepatic, and dense adhesions are usually present between the cyst and the stomach. Edlin[8] reports a case of pseudocyst of the pancreas extending from the superior border of the

pancreas through the lesser peritoneal sac through the esophageal hiatus into the posterior mediastinum between the parietal pericardium, aorta, and esophagus. The cyst contained 3,200 c.c. of clear, blood-tinged fluid. The pancreas was chronically inflamed with multiple cysts present. The patient had had recurrent attacks of acute pancreatitis for 10 years.

Clinical Aspects

The commonest symptoms of pseudocyst are pain, dyspepsia, weight loss, and jaundice.

Pain is the most common symptom, since pancreatitis is the most common cause of pseudocyst, and it is so often associated with the presence of a pseudocyst. The pain may be mild to very severe, and is usually epigastric in location. It may, however, be present in the right or left upper quadrants of the abdomen, or diffuse across the back, or through to the back at the level of the pancreas. The patient may bend forward in the knee-chest position in an effort to obtain relief. A sudden, severe pain may occur due to perforation of the cyst, a very serious complication when it does occur.

The *dyspepsia* includes anorexia, nausea, vomiting, etc., and is due to pressure on the adjacent structures, such as the stomach, duodenum, bowel, etc. *Weight loss* is secondary to the anorexia and vomiting. *Jaundice* is usually of the obstructive type, either due to pressure of the cyst on the common duct, or the associated pancreatitis or calculous disease of the biliary tract. In 13 of the 44 cases collected by Brilhart and Priestley[1] jaundice was present.

Physical Examination

Physical examination discloses the recent weight loss, often a palpable mass, and jaundice when present. The mass is usually found in the upper abdomen, and, in order of frequency, in the left upper quadrant, epigastrium, and least often in the right upper quadrant. The mass may only be palpable during an exacerbation, and may disappear between attacks. It may be soft or hard, tender or nontender, and usually does not move with respiration. The mass is often mistaken for malignancy. A palpable mass was found at operation in 32 of the 44 cases reported by Brilhart and Priestley[1].

Diagnosis

The diagnosis of pseudocyst is not usually made before operation. In only 9 of the 44 cases reported by Brilhart and Priestley[1] was the correct diagnosis made. The triad of upper abdominal pain characteristic of pancreatitis, pressure symptoms, and the presence of a palpable mass high in the abdomen, often tense and immobile, should make the examiner think of the diagnosis of pseudocyst.

Roentgen studies will help in establishing the diagnosis by demonstrating a soft tissue mass, or pressure phenomena or displacement of the stomach, duodenum, colon, or kidney. Calculi may be noted within the shadow of the pancreas on a flat plate of the abdomen. A nonfunctioning gallbladder is often present. Felsen, as quoted by Edlin[8], found that 21 per cent of patients with pseudocyst showed roentgen findings in the chest, including (1) elevated diaphragm, (2) disc atelectasis, (3) pleural fluid or thickening, (4) pneumonia, and (5) chylothorax.

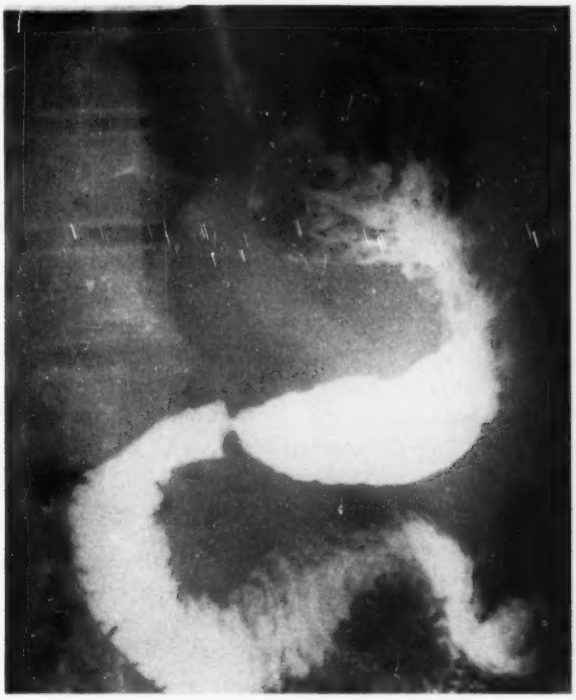

Fig. 1–Before operation.

Laboratory studies may show an elevated serum amylase, glycosuria, abnormal glucose tolerance curve, or other evidences of pancreatic insufficiency such as excess fat in the stools.

In the differential diagnosis, carcinoma of the pancreas, pancreatitis, cholelithiasis, abdominal tumors, or common duct stone must be considered. Gallbladder pathology is a frequent coincidental finding, and the complications of this disease may make the diagnosis difficult. Carcinoma of the body or tail of the pancreas may be impossible to differentiate except by surgical exploration.

Treatment

The treatment of pseudocyst of the pancreas is surgical. Conservative medical treatment is dangerous, since delay may lead to death from cachexia, or

more likely to rupture of the cyst into the free peritoneal cavity with resulting hemorrhage, shock and peritonitis. Koucky, as quoted by Brandenburg et al[4] found a 60 per cent mortality in a series of 6 cases treated medically.

Elman and Lieberman[10] report a case in which the mass disappeared spontaneously. The methods of treatment with surgery depend on the size and location of the pseudocyst. They are: (1) complete excision, (2) incision and marsupialization, (3) excision and partial pancreatectomy, and (4) incision and drainage, either internal or external.

Complete excision is the method of choice, but, because of the many adhesions to important structures usually found in association with the pseudocyst,

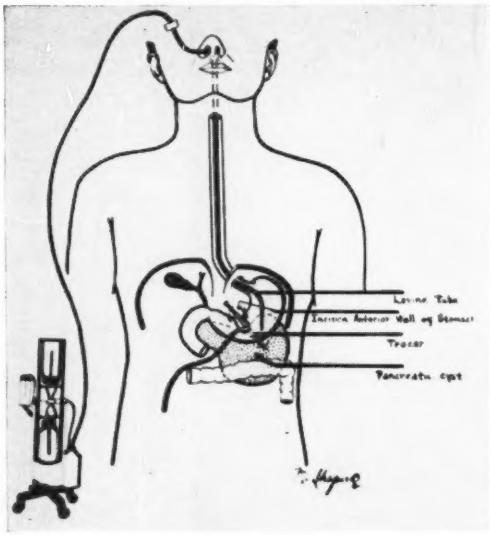

Fig. 2

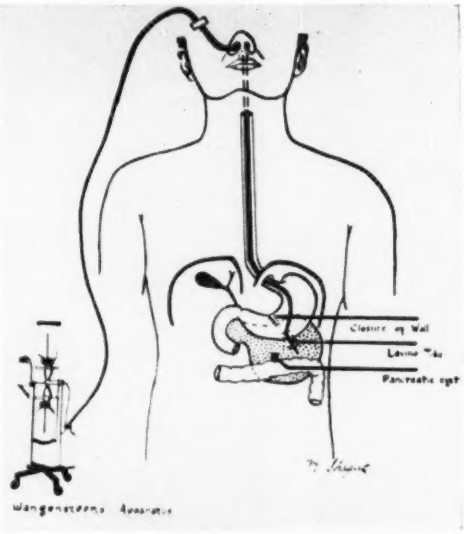

Fig. 3

Fig. 2–Trocar introduced into the cyst cavity.
Fig. 3–Levin tube sutured into cyst cavity.

it is rarely feasible. Brilhart and Priestley[1] were able to excise the cyst in 5 of their 44 cases.

Incision and marsupialization was first used as a method of treatment in 1882 by Gussenbauer. If, however, a duct leads into the cyst, treatment by this method usually leads to prolonged drainage, and formation of a chronic fistula with severe excoriations of the skin. Desvaux de Lyf[14] found that 80 per cent of 113 cases of pancreatic fistulae originated in cysts which had been so treated. Meyer et al[2] used this method in 21 of 27 cases, while Brilhart and Priestley[1] did this operation in only 12 of their 44 cases. Most authors believe that this method of treatment is best reserved for severely ill patients as a first stage preliminary to a subsequent resection.

Excision of the pseudocyst and partial pancreatectomy is best employed in cases involving the tail of the pancreas. Brilhart and Priestley attempted to do this in 2 cases involving the head of the pancreas, and had a fatal outcome in both cases.

Incision and drainage externally may be done when feasible. Internal drainage or cystovisceral anastomosis, however, is the treatment of choice in cases of pseudocyst which cannot be excised completely. The advantages of this method are (1) adequate drainage is secured, and (2) it permits the resorption of fluids, electrolytes, and enzymes into the gastrointestinal tract. The cyst may be anastomosed to the gallbladder or jejunum or stomach. The jejunum is the most available organ, and a Roux-Y type of anastomosis is used to prevent the regurgitation of intestinal contents into the cyst, and to allow pancreatic juice drainage into the jejunum where it normally belongs. This operation was first performed successfully by Hahn in 1927. In 1948 Gurwitz and Hurwitz applied the Roux-Y principle to the operation of cystojejunal anastomosis. Ryan and Murdock[9] believe this is the most suitable method of internal drainage. They review 12 cases of pseudocyst so treated and add a case of pseudocyst occurring in a 70 year old woman who had a 10 year history of recurrent pancreatitis. All 13 cases were treated successfully by cystojejunal Roux-Y type of anastomosis. Zaoussis[12] collected 105 cases of pseudocyst that were treated by internal drainage up to December 1951, 50 cases by cystogastric anastomosis, the rest by cystojejunal anastomosis. He reports 2 fatalities in the cystogastric group from hemorrhage, and 3 deaths in the other group (one from pulmonary embolism, and 2 from peritonitis).

In many cases of pseudocyst the cyst wall is markedly adherent to the stomach. In these cases the best method of internal drainage is the anastomosis of the cyst to the posterior surface of the stomach. This operation was performed for the first time by Jedlicka in 1923 after he had partially resected the cyst. Jurasz in 1931 was the first to perform the anastomosis without resecting the cyst. Zaoussis[12] was able to collect 50 cases of pseudocyst treated by cystogastric anastomosis in the literature up to 1952. There were 2 fatalities among the 50 cases, both due to hemorrhage. In this operation the transgastric approach is best since adhesions are minimal on the anterior aspect of the stomach, and there is less danger of leakage or hemorrhage. An incision is then made on the posterior wall of the stomach, and the lining of the cyst wall is approximated to the mucosa of the stomach. Dense adhesions will usually help to prevent early closure of the stoma. Zaoussis suggests keeping the patient prone for the first few postoperative days for this purpose. In the case reported below, the surgeon (A. B. Abrams) introduced a Levin tube, which had been placed in the stomach preoperatively, through the stoma into the cyst cavity so that continuous drainage of the cyst was maintained by attaching the Levin tube to Wangensteen suction.

Poer and Whitaker[5] report a case of pseudocyst which recurred 2 years after marsupialization. The cyst was then anastomosed to the stomach, and

the patient was well for the 9 years of observation. A barium meal study 6 years after this operation showed no barium entering the cyst, and no diabetes had developed. Elman and Lieberman[10] describe 2 cases. In one case, a large cyst developed in a 38-year old male 3 months after a severe attack of epigastric pain associated with a high serum amylase. The cyst was in the lesser sac, and it was successfully anastomosed to the greater curvature of the stomach. In their second case, a palpable mass, which appeared 4 months after repeated attacks of pancreatitis, disappeared spontaneously. Maxeiner and Maxeiner[11] report a case in a 38-year old man who developed a mass 5 inches in diameter

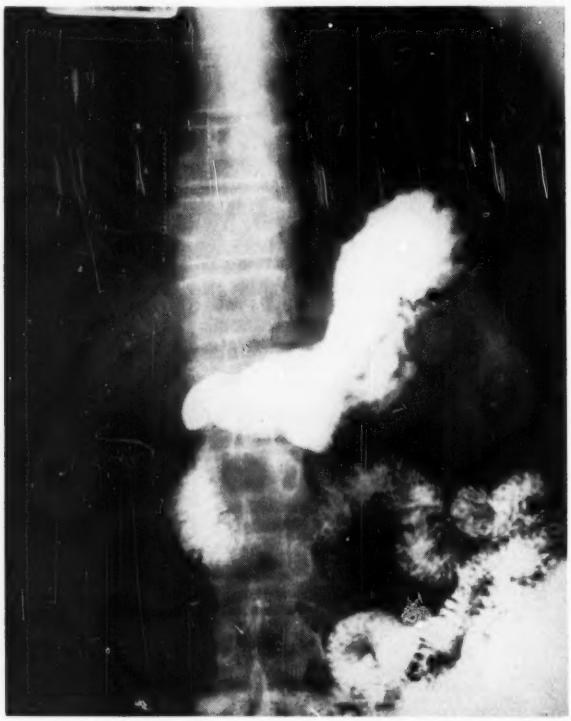

Fig. 4—Film taken five weeks after cystogastrostomy. No barium entered cyst cavity. Operative scar present on greater curvature.

3 months after an attack of pancreatitis. The cyst was anastomosed to the greater curvature of the stomach, and a cholecystectomy was done at the same time for gallstones. The patient had repeated hemorrhages, and 45 days later, at a second operation the cyst had disappeared, but a large ulcer was found on the posterior gastric wall. This was excised, but the hemorrhages recurred 4 times. Twenty days later a subtotal resection was done with a Witzel enterostomy for feeding. The patient then recovered, and is reported now well by the authors, but they conclude with a warning that such an ulcer complication must be considered in the event of hemorrhage after this operation for pseudocyst.

Brandenburg, Maddock and Schweitzer[4] report a case anastomosed to the posterior wall of the stomach after evacuating 300 c.c. of fluid from the cyst. On the sixth postoperative day an oral barium meal did not enter the cyst. A roentgen study 8 months later showed the operative defect, but no barium entered the cyst. Gastroscopy revealed a normal stomach except for slight injection of the mucosa, and a patent stoma was visualized on the posterior wall connecting to the cyst. There was no diastase in the gastric fluid. A 2 year follow-up study revealed a well patient despite an irregular diet and severe alcoholism. Zaoussis[12] found the operation of cystogastrotomy successful in 33 of 45 cases that reported a follow-up study.

The theoretical objection to this operation of cystogastric anastomosis has been disproved, namely that gastric contents will enter the cyst cavity and cause necrosis and infection, or that pancreatic ferments will injure the stomach mucosa. Rössing[15] believes that intraabdominal pressure and the flow of pancreatic juice prevents the reflux of gastric juice into the cyst cavity. Altshuler attempted to pass barium through the stoma by different positioning of the patient with no success. Adams, Chesterman, Josa, Poer, Vecchi, Kunc, and Brandenburg et al[4] did not find barium in the cyst cavity in postoperative roentgen studies. In the case reported below, an oral barium meal study 5 weeks after the cystogastric anastomosis showed no barium entering the cyst cavity. In our opinion, just as in a posterior gastroenterostomy with a functioning, nonobstructed pylorus, the food or a barium meal will tend to go through the normal pathway of the pyloric canal rather than the enterostomy stoma, just as after a cystogastric anastomosis the gastric contents will pass through an unobstructed pylorus and not enter the cyst cavity. Experimental work has also shown that pancreatic secretions pass into the stomach, but they are innocuous. Grey in 1917, in experiments on dogs, anastomosed a large pancreatic duct into the stomach wall, and ligated a lesser duct. Alkaline fluid passed into the stomach, but no damage was done. Tripodi[6] in 1939 implanted the tail of the pancreas into the stomach. Necropsy 4 weeks later showed no erosions, and no digestion of the walls of the stomach. Pancreatic functions were not impaired by this experimental anastomosis.

Postoperative Treatment and Course

Wangensteen suction is used postoperatively plus intravenous fluids commensurate with the electrolytic balance studies. Annis and Hallenbeck[7], in studying the effects of Banthine on pancreatic secretion, show that in small doses Banthine is anticholinergic, in moderate doses produces a ganglionic-blocking effect, and in large doses a curare-like effect. Also, pancreatic secretion depends on gastric secretion, digestion in the stomach, and the vagal innervation of the pancreas. Since Banthine reduces gastric secretion and gastric motility through its effect on the vagus nerve, it is probable that in adequate doses it would decrease pancreatic secretions. A dose of 100 milligrams has

been advised. In our case, however, Banthine was used for a short time with no notable change in the amount of pancreatic secretion.

Manifestations of pancreatic insufficiency may follow the operation, with symptoms of fat intolerance and occasionally nausea and vomiting. This is relieved by a high protein, low fat diet plus full doses of pancreatic extract.

Case Report

J. H., white, married male, age 52, came to me (S. M. G.) on March 26, 1952 complaining of severe pain in the epigastrium, radiating to the right upper quadrant. For over 10 years he had had an intolerance to fatty foods, plus heartburn and belching. A diagnosis of peptic ulcer or gallbladder disease had been made on various occasions during these 10 years. He had frequent attacks of pain during these 10 years, each attack usually lasting four to five hours. A low fat diet gave some relief. On March 22nd he developed this constant, severe epigastric pain, temperature of 100°, and dark urine was noted by the patient. Physical examination revealed a slight icteric tinge in the sclerae, and marked tenderness and rigidity in the right upper quadrant of the abdomen. No organs or masses were palpable. A blood count showed the hemoglobin to be 13.5 grams, and the white count 7.650 with a normal differential. The urine contained bile pigment. Because of the previous diagnoses of ulcer and/or gallbladder, it was decided to do a partial roentgen study. A flat plate of the abdomen in the upright position did not show air under the diaphragm. Examination of the biliary tract after a double dose of Priodax tablets failed to visualize the gallbladder, and no radio-opaque calculi were noted. An oral barium meal study was then done on the same morning, and no ulcer was found in the stomach or duodenum. The duodenal bulb and duodenal curve did not show evidence of pressure (Fig. 1).

He was sent to the Newark Beth Israel Hospital with a diagnosis of acute cholecystitis, and surgical exploration was advised. Operation on March 27th, 1952 by Dr. A. B. Abrams revealed a large, thickened gallbladder filled with stones. A large amount of serosanguinous fluid was present in the abdomen. The pancreas appeared markedly edematous, and there was evidence of pancreatic enzyme reaction immediately adjacent to the duodenum. The stomach was adherent to the pancreas, but there was no evidence of intrinsic pathology in the stomach or duodenum. Because of the presence of the acute pancreatitis plus the gallstones, it was decided to do a cholecystostomy after the removal of some 60 stones from the gallbladder. A large tube was inserted in the gallbladder for drainage. The patient had no pain or complications, and on the 10th day the drainage tube fell out. He then began to complain of anorexia, heartburn, and epigastric pain. His temperature, which had been normal for 10 days, now rose to 101°. A blood count done on April 10th disclosed a hemoglobin of 93 per cent, red count of 4.4 million, and a white count of

26,000 with 79 per cent polys and 9 per cent stabs. On April 12th, 16 days postoperative, he had a chill, and his temperature rose to 105°. Penicillin and streptomycin were given, but the temperature continued to range from 100° to 102° daily. Serum amylase studies on April 15th and April 18th revealed increased amylase in the blood. On April 22nd, four weeks after operation, a mass was palpable in the left upper quadrant of the abdomen. It did not move with respiration, but was moderately tender to palpation. Blood sudies revealed a urea nitrogen of 11 mg. per 100 c.c., blood protein 6.4 per cent, serum bilirubin 0.27 mg/100 c.c. with the van den Bergh reported as a direct delayed reaction. The blood sugar was 113 mg./100 c.c., alkaline phosphatase 5.1 Bodansky units. The mass became progressively larger daily, and more tender. On April 28th, or 37 days after the first operation, surgical exploration was performed by Dr. A. B. Abrams under spinal and sodium pentothal anesthesia. At operation, a mass the size of a grapefruit was found between the body of the pancreas and the posterior aspect of the stomach. The gastrocolic omentum was opened and an anterior gastrostomy was done. A trocar was forced through the posterior wall of the stomach into the cystic mass (Fig. 2) and about two liters of milky fluid, high in amylase content, were aspirated. The stoma was then slightly enlarged, and a Levin tube, which had been placed in the stomach preoperatively, was then introduced into the cyst cavity through the stoma and sutured in place (Fig. 3). The stomach opening was then closed, and the abdomen closed in the routine manner. Postoperatively, the Levin tube was attached to a Wangensteen suction. There was considerable drainage of fluid through the Levin tube, with the drainage strongly positive for amylase until May 4th, or 13 days postoperative, when the drainage was found to contain only hydrochloric acid. Since the tube was now apparently in the stomach it was removed. The patient developed an atelectasis of the left lower lobe on the fifth postoperative day, but this cleared in five days. On May 7th, or nine days after the operation, all blood studies were normal. The patient was discharged on May 11, 1952, or 14 days after the cystogastrostomy, with no symptoms, a normal chest roentgenogram, normal blood count and normal blood chemistry, and the wound entirely healed. He was placed on a low fat, bland diet. He has been entirely well, free from gastric symptoms, up to the present time, or 30 months. A barium meal study on June 2, 1952 revealed a normal stomach and duodenum except for the operative scar on the greater curvature of the stomach corresponding to the site of the stoma into the cyst cavity. No barium was seen to enter the cyst cavity at any time during the fluoroscopic or roentgen study (Fig. 4).

Conclusions

In conclusion, we present a case of pseudocyst of the pancreas, retrogastric in position, occurring after ten years of recurrent, unrecognized attacks of pancreatitis. It is the 51st case recorded in the literature apparently cured by the internal drainage method of cystogastrostomy. The use of the Levin tube sutured

into the cyst cavity through a stoma made in the posterior wall of the stomach is suggested as an aid to successful drainage.

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A RARE CASE OF CONGENITAL MICROCOLON

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The following case of congenital microcolon is presented because of its extreme rarity. Statistically speaking one or less cases in 500,000 autopsies may be found.

None of the many theories as to origin of intestinal atresia has been proven. Atresia may frequently occur in different parts of the gastrointestinal tract.

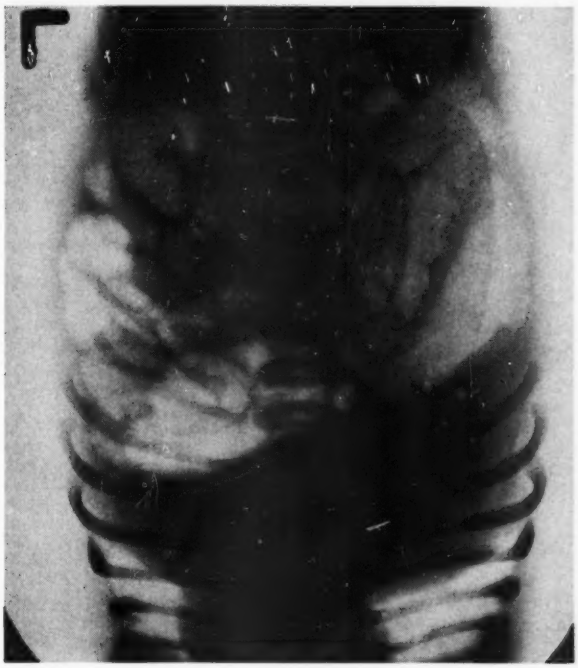

Fig. 1

An extreme narrowing of the entire colon with or without narrowing of the lower ileum and always without any atresia of the rectum has been named congenital microcolon and described as occurring as an isolated malformation. It is not an atresia *per se*, meaning a substitution of fibrous tissue for bowel, but more of an *en miniature* edition of a normally developed colon.

The interesting feature of our case is, that it does represent such an isolated malformation without any other atresia higher up.

Case Report

Case 54-1609:—Kew Gardens General Hospital. The baby was born March 9, 1954 at 8 A.M. The mother is 28 years old, healthy, G 2, P 2, group A.RH+. The pregnancy was normal, the delivery without difficulties. No abnormalities, especially of anus and/or genital organs were noticed at birth. Less than 24 hours after birth the baby developed a mild jaundice. A complete bloodstudy on the second morning of life did not reveal any abnormalities in the red cells, a total white count of 25,000 with 84 per cent polys was found. At that time a periumbilical redness was noted and penicillin given. The baby so far had taken her nourishment very well.

In the late evening of the second day the baby started to vomit profusely, the vomitus being gastric content without meconium. The abdomen at that

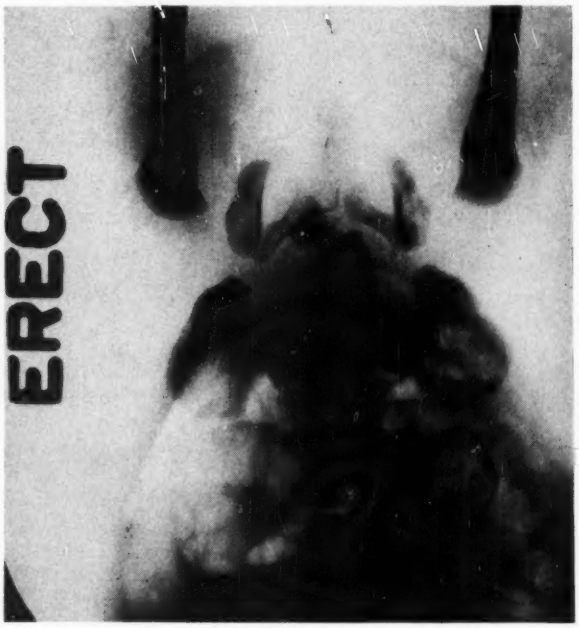

Fig. 2

time was distended and tympanitic, the abdominal veins were enlarged. Upon rectal examination the anal region was found to be normally developed, the examining finger and subsequently the proctoscope could not be extended above 5 cm. from the external anal region. A flat plate of the abdomen and a barium colon enema was done on the third morning. (Drs. M. Fuhrman and J. Sumers). Examination of the flat plate shows considerable gaseous distention of stomach, small and large bowel. B.A. examination of the large bowel by means of a barium enema shows the presence of a partial obstruction approximately 5 cm. proximal to the anus at the junction of the rectosigmoid and rectum. After

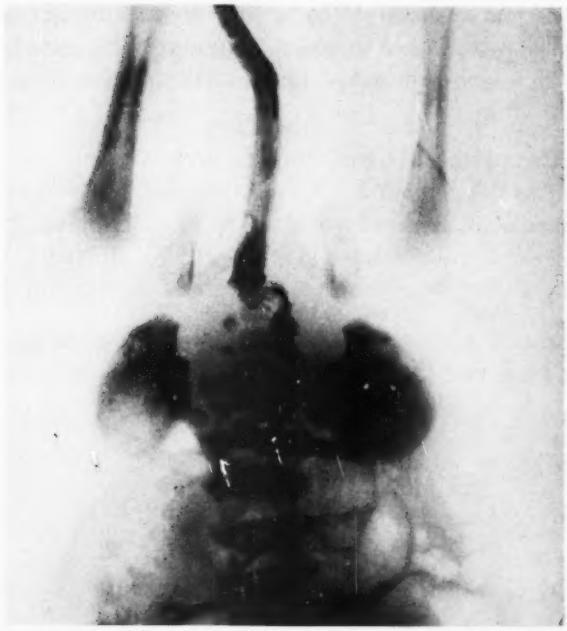

Fig. 3

Fig. 4

repeated attempts and overdistention of the rectal ampulla, a small quantity of barium passed beyond the obstruction. The narrowing noted is approximately 2 cm. in length. The bowel proximal to the side of obstruction is diminished in calibre.

A further attempt to pass the stricture with a small size rubber catheter was unsuccessful. As the general condition of the baby, despite use of antibiotics, clysis, blood transfusions, became worse, it was decided, after frequent pediatric (Dr. P. Chasin) and gastrointestinal (Dr. A. X. Rossien) consultations, to operate, the preoperative diagnosis being intestinal obstruction lower sigmoid.

Operation (Dr. S. Klein):—The abdomen was opened through a transverse incision just above the umbilicus. The stomach, duodenum and proximal jejunum

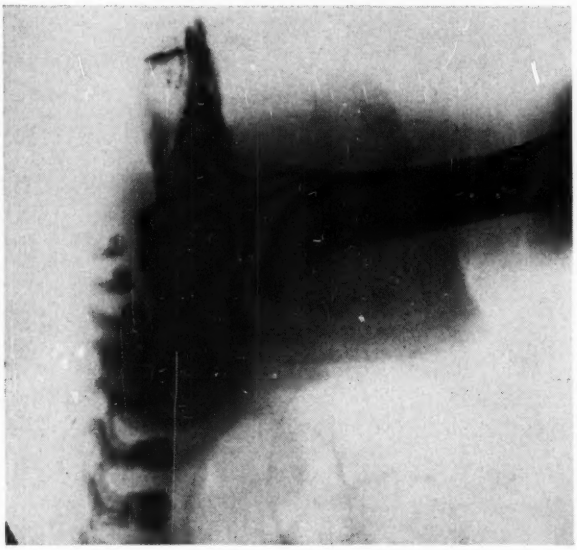

Fig. 5

were greatly distended. The distal part of the jejunum and the proximal part of the ileum were trapped in a pocket of omentum, the ileum part being gangrenous with a perforation through which meconium was pouring. The ileum distal to the gangrenous part seemed very narrow, the entire colon appeared as a cord-like structure, the appendix was present, but very small. The perforated area of bowel was exteriorized and the abdominal wound closed. The postoperative prognosis was very poor. The baby expired 93 hours after birth.

Summary of pathological findings (Drs. Louis Odenky and Julius Rosenthal) Nu. A-54-8.

Gross Examination:—The body was that of a female infant 47 cm. in length and approximately 7 lbs. in weight. The infant was well formed and showed no

congenital abnormalities on the surface of the body. The heart was of average appearance. The posterior portion of both lower lobes of the lungs showed congestion. Genitourinary tract, liver, pancreas, spleen, adrenals and brain showed no marked gross abnormalities.

The principal pathology was in the abdomen. There was a transverse supraumbilical incision about 12 cm. long, through which a hemorrhagic loop of intestine protruded. Further examination revealed this loop to be part of the jejunum which was dilated, hemorrhagic and gangrenous. The involved jejunum merged with the ileum, which had a similar appearance. There was a perforation about 1 cm. in length at about the junction of the lower and middle thirds of the ileum. It was associated with the presence of a considerable amount of fecal-like material in the abdominal cavity. The caliber of the jejunum

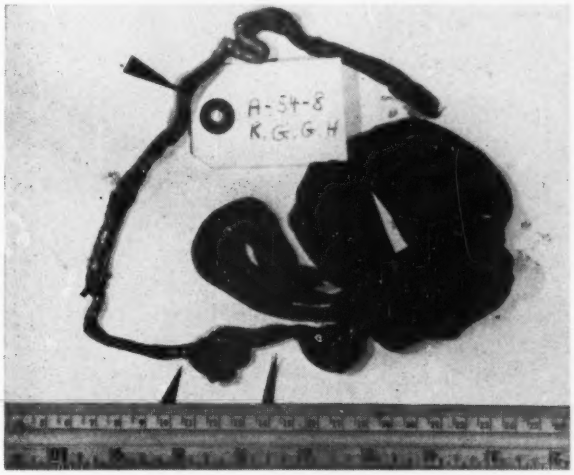

Fig. 6

and ileum varied from approximately 1.5 to 3 cm. The peritoneum generally showed considerable congestion. The colon was small and narrow. Its length was 18 cm. Its width was approximately 0.6 cm. The width of the lumen was approximately 0.1 to 0.3 cm. The organ also had a greyish-white contracted appearance. On section the lumen was found to be markedly narrowed, but an opening was traced from the cecum to the anus. The lumen contained viscid greyish, partly gelatinous material. No meconium was found in the colon. The appendix was 4 cm. in length and approximately 0.3 cm. in width. The lungs, liver and kidney showed a moderate degree of congestion.

Microscopic Examination:—Jejunum. Only portions of the mucosa were found to be intact. The remainder of the wall had an edematous, hemorrhagic appearance. Many of the small blood vessels appeared widely dilated. The

muscularis had in addition to the hemorrhagic infiltration also scattered polymorphonuclears.

Colon. The architecture of the colon was clearly discernible. The lumen was present and contained a mass of amorphous material. The mucosa, submucosa and muscularis were well defined, and showed no marked abnormalities other than that all the layers were proportionately narrow. The serosa showed considerable congestion, some hemorrhage and scattered inflammatory cells. Another section of colon showed some edema of the wall as well as the presence of dilated vessels and some hemorrhage.

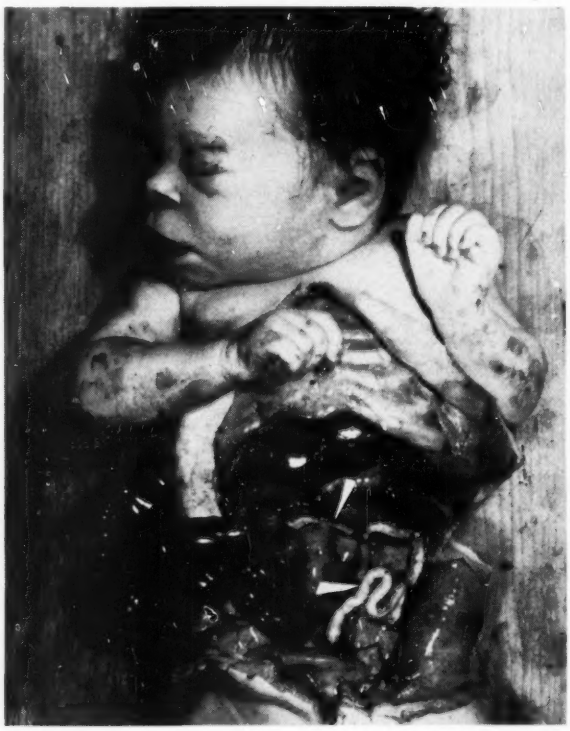

Fig. 7

PATHOLOGICAL DIAGNOSIS

1. Congenital malformation of the entire colon with marked narrowing of the lumen-microcolon.

2. Congestion, gangrene of jejunum and proximal ileum and perforation in the ileum.

3. Beginning generalized peritonitis.

SUMMARY

A rare case of congenital microcolon has been presented. Of the very few cases published in the world-literature during the last 50 years the majority were connected with atresia and/or other congenital deformities.

The fact that our case represents a classical picture of an isolated malformation has been emphasized.

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President's Message

With the First Convention of the American College of Gastroenterology behind us many new names and faces have entered the scene, all a part of a bigger, better, and more capable national organization. We hope we have pleased our new friends, and we hope that they will join us in our effort to improve and promote better gastroenterology.

Your old Officers and Trustees, under the leadership of your immediate past President and his predecessors, have worked very hard to perfect the Constitution and By-laws by which we operate. Their trial run was a splendid success, although everyone realizes that only time and experience will smooth out the rough spots.

We must now consolidate our gains, and develop our resources. Here is where the Governors can be of the greatest help. This is your College, and it is hoped that your suggestions and unselfish assistance will serve to create and maintain the lofty principles for which we are banded together.

Our effort must first be in the direction of good medicine, and next in the direction of good gastroenterology and its allied special fields. This means the enlisting, developing and indoctrinating of broad, sound men in our College. We have them, and will attract others if we remain humble and work together for the common good.

The 1955 convention program is in the process of formation under the able leadership of Dr. Arthur A. Kirchner, of Los Angeles. The Postgraduate course is being thoroughly thought out and re-organized for next year by Dr. C. Wilmer Wirts, of Philadelphia. Let's give them any help we can, wherever and whenever they may need it.

Dr. John E. Cox was charged with the responsibility of putting on the second regional meeting, to be held in the city of Memphis. He has informed us that arrangements have been made with the Hotel Peabody for Sunday, 24 April 1955. More details will be forthcoming at a later date. Let's all plan to go to Memphis, in April.

There is much work to do, and many hands will make it easy.

EDITORIAL

THE GASTROENTEROLOGIST IN ARMY MEDICINE*

SILAS B. HAYS, MAJOR-GENERAL, U.S.A.†

Washington, D. C.

I am particularly honored for the privilege of speaking before the First Convention of the American College of Gastroenterology. Your president, Doctor Sigurd W. Johnsen, and his staff in the College should be congratulated on establishing the College policies of: (1) New and higher standards of membership and advancement in the College; (2) The establishment of a postgraduate course and (3) A research program.

As Deputy Surgeon General of The Army, I am keenly interested in the field of gastroenterology because of the outstanding contributions made by Army surgeons in this field of medicine.

The story of Army Surgeon William Beaumont and his fistulous patient, Alexis St. Martin, is a classic in American Medical History and physiologic research. The famous incident occurred on 6 June 1822 in the American Fur Company Store, when 19 year old Alexis St. Martin, a French Canadian Voyageur, received the accidental discharge of a shotgun in the abdomen. The story of Beaumont's surgical care of his tempermental patient and his subsequent studies of the physiology of digestion, by way of St. Martin's gastric fistula, is familiar to all students of medical history. Alexis St. Martin died in 1880 at an advanced age. It is noteworthy that there is a newly completed memorial to Beaumont on Mackinac Island, Michigan, to commemorate the 100th anniversary of his death in 1853.

Typhoid Fever

Another contribution made in the field of gastroenterology has been the control of typhoid fever in our Armed Forces. Typhoid fever was not accurately reported in the United States before 1862, because it was confused with dengue and malaria. From 1862 to 30 June 1866, however, over 79,000 cases of typhoid fever, with over 29,000 deaths, were reported to have occurred in the Federal Troops. In the Spanish American War, approximately 110,000 troops were mobilized. Over 20,000 cases of typhoid fever occurred in military camps in the United States with 50 per cent of *all* deaths in the military being due to typhoid fever alone. A medical board was appointed by The Surgeon General, O'Reilly, in 1908 to study the merits of antityphoid vaccination. Captain Frederick Fuller Russell was a member of this Board. After 3 years of study and the inoculation

*Address delivered at the Annual Convocation of the American College of Gastroenterology, Washington, D. C., 25 October 1954.

†Deputy Surgeon General, Department of the Army.

of volunteers, the United States Army adopted compulsory prophylaxis against typhoid fever. Shortly thereafter, Captain Russell inoculated 20,000 troops, mobilized on the Mexican Border, and the only case of typhoid fever occurring in camp was that of a nonvaccinated teamster. In 1910, Brig. General Carl Rogers Darnall first used liquid chlorine to purify drinking water. A continuous low incidence rate of typhoid fever occurred in the United States Army for the first time as a result of the above developments. Therefore, during World War I, our Army had the low morbidity rate of 0.37 per 1,000 mean strength per year, as compared to 112,000 and 170,000 cases each, for the German and Austrian Armies respectively, who had no compulsory typhoid fever immunization. During World War II, from 1942 to 1945, there occurred only 505 cases of typhoid fever with 35 deaths in our entire Army.

Bacillary Dysentery

Another contribution was in the control of bacillary dysentery. This disease plagued our Army in all its wars before World War I. One of the important early accounts of bacillary dysentery in 1804, was by Army Surgeon James Mann for which he was awarded the Boyston Prize in 1806. The dysentery bacillus occurs in several varieties, each of which is known by the name of its discoverer. Three of them, Flexner, His-Russell and Strong, bear the names of officers who held commissions in the Regular Army, i.e., Colonel Simon Flexner, Brig. General Frederick F. Russell and Colonel Richard Pearson Strong. During World War II, from 1942 to 1945, approximately 26,000 cases of bacillary dysentery, with only 16 deaths were reported in our entire Army.

Amebic Dysentery

Over 34,000 cases of amebiasis occurred in our troops from 1942 to 1945; over 22,000 of these alone were reported during 1945. Amebic dysentery is from the standpoint of incidence, by far, the most important of the dysenteries. During 6 of the past 7 years, amebic dystentery has accounted for 50 per cent or more of all dysentery reported among United States Army troops world-wide. The high incidence of bacillary and amebic dysentery occurring in the United Nations' prisoners-of-war has facilitated the study of these important diseases. Based upon studies on 1,408 patients in the United Nations' prisoner-of-war camps in Korea from whom pathogenic shigellae were isolated, it was found that Terramycin, Aureomycin and Chloramphenicol were effective in treating *sulfonamide resistant shigellosis.* The responses to therapy of 538 cases of uncomplicated acute amebic dysentery in the United Nations' prisoner-of-war camps in Korea were studied to determine the comparative efficacy of amebacides and antibiotics used alone and in combination. The results of these studies were excellent. *Such an investigation with our present incidence rate, in the United States, would take many years to duplicate.*

Food Poisoning

There is a *low* incidence of bacterial food poisoning in the Army. During 1953, the incidence of bacterial food poisoning (food infection) averaged 0.63 per 1,000 mean strength for 1953. This is the lowest level for this condition, since the early phase of the Korean Campaign, when the incidence rate in July 1951 reached 2.53 per 1,000 average strength per year. Outbreaks of food poisoning in Army messes is not a matter to be condoned, as those of you who have served as medical officers during your Army service well know. Yet, food poisoning reports in the Army are a matter which cannot be compared to civilian medicine, since in the latter, we are only beginning to acquire incidence data. The Armed Forces Epidemiological Board, Commission on Enteric Infections, is currently carrying out studies of the different pathogens of enteric diseases, as well as studies on viruses that may well cause some of these food poisoning outbreaks, or, be involved in the pathogenesis of bacterial infections.

Schistosomiasis

With the landing of our troops in Leyte and other areas in the Southwest Pacific during the period of 1944 to 1945, 1,544 cases of schistosomiasis occurred with only 1 death reported. Preventing our military troops from bathing in fresh water streams infested with infected snails, resulted in control of the schistosomiasis problem in our Armed Forces in that area. Treatment to date for *Schistosomiasis Japonicum*, however, is still far from satisfactory, and we are searching for a drug to effectively treat this disease. Studies are being conducted to develop a *protective ointment* against cercariae producing schistosomiasis in man; also, studies of the epidemiology of schistosomiasis are in progress at the Army Tropical Medical Research Laboratory in Puerto Rico in conjunction with the University of Puerto Rico.

Infectious Hepatitis

In 1942, our medical officers were suddenly confronted with a serious medical problem, namely, infectious hepatitis. Prior to this time, we had encountered an occasional case of so-called catarrhal jaundice in our Army posts. Now, suddenly, thousands of our troops were incapacitated with this disease. The combined efforts of our medical officers and civilian consultants gradually cleared the mystery of infectious hepatitis. Tremendous progress has been made in the medical management of this disease since World War II. Recent studies now show that *early* and *controlled progressive ambulation* is not harmful to the hepatitis patient. I do not intend to minimize the exemplary assistance and effort given the Medical Services of the Armed Forces by our civilian colleagues during World War II and the Korean War. Only by joint cooperation between medical officers and civilian consultants, have improved methods in the diagnosis and treatment of disease been developed in so short a time since World War II.

Peptic Ulcer

Comment has been expressed from several quarters and from our civilian medical consultants, that there is a high incidence of peptic ulcer occurring in the members of the Armed Forces. At first impression, this appeared to be true, because in the military it is necessary to treat these patients in a hospital until the ulcer is healed. When, however, one examines the statistics of the hospital admissions for peptic ulcer during the war years 1942 to 1945 and again from 1950 through 1952, we find the incidence to be less than 3 hospital admissions per 1,000 mean strength per year. The real problem, however, is the number of peptic ulcer patients who require *separation* from military service because their ulcers "*just won't heal*" despite the best of treatment afforded these patients. We also realized that the potential ulcer patient is most susceptible to the new stresses and emotional conflicts encountered after he has been drafted into military service. With this viewpoint in mind, we have further developed the policy of better doctor-patient relationship in our hospitals. In addition, ward medical officers conduct group psychotherapy for these patients, by frequent informal discussions on their wards; the use of blackboard diagrams describing their disease, and explaining the importance of regularity of diet, medication and rest. Individuals with emotional conflicts are referred to the psychiatrist for therapy; those with personal problems, are cared for by the Social Service or Red Cross worker. Despite all this *special* treatment, the period of hospitalization continues to be long and the same high discharge rate continues. The noneffective rate and large loss of manpower in this group is a problem, that we in the military, hope our civilian colleagues can help us solve, for the success of any military mission is greatly dependent on maintaining the good health of our fighting forces.

The history of medical progress made in the United States Army Medical Corps clearly demonstrates that the role of the gastroenterologist in Army medicine is a very important and continuous one.

The ultimate goal in this field of medicine can be attained only by the integrated teamwork of the various specialists in medicine; each contributing his part to assist us in maintaining and preserving the health of our troops in the Armed Forces. We sincerely hope, as has been true in the past, that the exemplary cooperation between the military and civilian physicians will continue.

ABSTRACTS FOR GASTROENTEROLOGISTS

LIVER AND BILIARY TRACT

HEPATITIS OF MALARIAL ORIGIN: Arthur E. McMahon, Jr., James E. Kelsey and Donald E. DeRauf. A.M.A. Arch. Int. Med., 93:379, (March), 1954.

Fifty-four Korean war veterans suffering from tertian malaria had peritoneoscopic liver biopsies and various liver function tests before and after institution of treatment. All showed signs of acute hepatitis. The liver biopsies revealed degenerative changes of the liver cells, mononuclear cells, infiltration of sinusoids, increased activity of Kupffer cells and occasional derangement of hepatic architecture. The foremost abnormalities of liver function were found in cephalin flocculation, thymol turbidity, B.S.P. excretion and ratio of total to esterified cholesterol. Clinical symptomatology of hepatic disease, especially enlargement and tenderness of the liver, was present only in half of the cases. All hepatic abnormalities were found before administration of the antimalarials and disappeared gradually while the patients were under treatment. Thus, the specific antiparasitic drugs did not seem to have any adverse effect on the hepatitis, however, it appeared advisable to add a specific regimen for the treatment of the liver disorder.

H. B. EISENSTADT

INTRAHEPATIC CHOLANGEOJEJUNOSTOMY FOR BILIARY OBSTRUCTION: H. N. Lipman and W. P. Longmere. Surg. Gynec. & Obst., pp. 363-372, (March), 1954.

Five cases are reported in which an intrahepatic cholangeojejunostomy with partial hepatectomy was employed for the relief of extensive extrahepatic biliary obstruction bringing to 23 the total number of cases reported to date. The procedure has been employed by the authors for extensive benign obstruction of the extrahepatic biliary system when the usual methods of re-establishing biliary enteric continuity have proven to be inadequate. The authors emphasize that the operation is not to be reserved until biliary cirrhosis and its attendant sequelae have occurred, but rather it should be utilized in its logical sequence in the event that other perhaps more physiologic procedures have proved to be unsuccessful. The authors in addition conclude that operative cholangeography is an invaluable adjunct in the performance of intrahepatic cholangeojejunostomy.

J. R. VAN DYNE

POSTCHOLECYSTECTOMY ORAL CHOLANGIOGRAPHY: A Preliminary Report; J. R. Twiss, S. A. Beranbaum, A. Gillette and M. H. Poppel. Am. J. M. Sc. pp: 372-386, (April), 1954.

The authors use the following technic: The patient is instructed to take 6 tablets of Telepaque at 5 minute intervals beginning at 6 P.M. After the last tablet one teaspoon of paregoric is given. The total amount of water given is not to exceed ½ glass. At 11 P.M. the above procedure is repeated. At 8 A.M. one teaspoon of paregoric is again given with a swallow of water. At 10 A.M. P-A and oblique films are taken localized to the right upper quadrant. If the bile ducts are visualized, an inhalation of amyl nitrite

is given with the patient in the recumbent posture. Additional films are taken at 15 and 30 minutes. If there is no emptying of the bile ducts, a fat meal is given and another film is taken in 30 minutes. The lack of response to amyl nitrite is presumptive evidence of organic obstruction, of the ampulla of Vater, or the common bile duct sphincter.

The authors believe that the method is indicated in the postcholecystectomy syndrome and in cases of nonvisualization of the gallbladder. In the cases studied to date, 85 per cent of the postcholecystectomy patients had visualization of the bile ducts. With biliary dyskinesia, about 70 per cent of the bile ducts visualized were found to be dilated. In postcholecystectomy patients without symptoms, none of the bile ducts were significantly dilated. In addition the authors found that pathologic conditions such as cystic duct remnants could be detected by this method.

J. R. VAN DYNE

INTRAVENOUS CHOLECYSTOGRAPHY AND CHOLANGIOGRAPHY; PRELIMINARY OBSERVATIONS: J. E. Berk, R. E. Karnofsky, H. Shay and H. M. Stauffer. Am. J. M. Sc. pp. 361-371, (April), 1954.

The authors present preliminary observations on rapidly obtained cholecystography and cholangiography, using a new intravenously injected medium currently designated Be-426 (Cholegrafin-E.R. Squibb Co.; and Biligrafin, Schering & Co.). It was found that the bile ducts are visualized before the gallbladder becomes opaque, and may be detected 10 minutes after intravenous injection of the medium. Visualization of the gallbladder by this method is of a quality comparable to that obtained after the newer orally administered cholecystographic media. Radiolucent calculi are readily detectable and clearly shown. The common duct and sections of the major extrahepatic biliary tree may be visualized with a high degree of regularity in patients after cholecystectomy. Impairment in liver function interferes with the secretion of Be-426 into the bile, and there may result faint or no visualization of the ducts and gallbladder. The degree of opacification of the bile ducts is sometimes faint and the terminal end of the common bile duct may frequently be poorly seen. In this study no serious local or systemic reactions occurred and it is concluded that this new method may be a valuable adjunct.

J. R. VAN DYNE

HEPATIC CIRRHOSIS OF PERITONEAL ORIGIN: Jean Olmer, E. Gascard and P. Casanova. Presse Medicale. 62:600-602, (April 21), 1954.

The authors call attention to the part of certain chronic peritonitis of undetermined etiology in the origin of cirrhosis of the liver. Such an etiology has been long ago described as falling in the group of cirrhoses due to tuberculous peritonitis. But in many cases the tuberculous nature of the peritoneal infection has been unquestionably excluded. It is related to a process developing in the liver, equivalent to that described by Chabrol as present in the spleen and achieving Banti's syndromes of peritoneal origin.

A case is reported in detail which is typical of that cirrhosis of the liver originating from the peritoneum. The disease broke out with massive hematemesis followed by rapid development of an ascitoedematous syndrome. Examination showed an enlarged liver, voluminous esophageal varices and there was unquestionably absence of initial splenomegaly (operative findings). The evolution, which from the onset seemed to proceed from a very severe form of portal hypertension syndrome, has been controlled by a portacaval anastomosis.

At operation was seen a "gangue" of perihepatitis suggesting Carchman's "iced liver". No specific change was histologically demonstrated, but only a fresh evolutive cirrhosis with annular development tendency.

The performance of the portacaval anastomosis has been very successful in spite of the postoperative appearance of an enlarged spleen.

Thus should a peritoneal etiology be suspected in those cases of hepative cirrhosis the origin of which remains unrecognizable.

GUY ALBOT

DYSTROPHIA MYOTONICA WITH ASSOCIATED SPRUE-LIKE SYMPTOMS: K. K. Kaufman and E. W. Heckert. Am. J. Med., 16:614, (April), 1954.

A case of dystrophic myotonia associated with sprue-like symptoms was observed. The diagnosis was based upon progressive muscular atrophy of face, neck and upper extremities with myotonic reaction (delay of muscular relaxation). There were also presenile cataracts and gonadal failure (testicular atrophy, sparse axillary and pubic hair and decreased urinary 17-ketosteroids). The patient had persistent steatorrhea, nausea, vomiting, diarrhea, flat glucose and Vitamin A absorption curves and severe weight loss. The intestinal symptomatology was attributed to the atrophy of the smooth muscles of the intestines. This case illustrates the need to consider various myopathies in the differential diagnosis of the sprue syndrome.

H. B. Eisenstadt

ROENTGEN THERAPY OF HEPATIC METASTASES: R. Phillips, D. A. Karnofsky, L. D. Hamilton and S. C. Nickson. Am. J. Roentgen. 71:826-834, (May), 1954.

The results of treatment of hepatic metastases, secondary to carcinoma of the breast, bronchus, and gastrointestinal tract are reported in 36 patients; symptomatic improvement was obtained in 26 patients. The symptomatic relief of pain, anorexia, nausea, vomiting, weakness, fatigue, sweating and abdominal distention was accompanied by a reduction in the size of the enlarged liver, by gain in body weight (not fluid), and an improvement in liver function as measured by determination of the serum bilirubin, alkaline phosphatase, cholesterols, proteins, bromosulphalein, prothrombin time, cephalin flocculation and thymol turbidity tests. There was no evidence of liver damaged at the radiation dose levels used.

J. R. Van Dyne

VIRAL HEPATITIS. PROBLEMS AND PROGRESS FOR 1954: S. R. Neefe. Am. J. Med. 16:710-728, (May), 1954.

This is a splendid review and presents current information regarding hepatitis due to infection by Virus I.H., and Virus S.H. Etiology, epidemiology, immunology, clinical manifestations and complications, laboratory findings, the carrier state, diagnosis, prevention, and treatment are all discussed. Of special interest is the difficult analysis of the unduly protracted course and the revision of views as to early ambulation. Patients are urged to stay in bed as long as acute symptoms persist. Once they begin to feel well, regardless of the degree of jaundice, they should not be forced to stay in bed more than an hour after each meal. Restriction to the hospital ward, however, was essential to decrease undue activity or exertion. Patients whose bromsulfalein retention stabilized between 5 and 10 per cent (for 45 minutes) were discharged from the hospital with safety. They were then followed weekly for 2 weeks with physical reexamination, serum bilirubin and bromsulfalein tests.

J. R. Van Dyne

OCCURRENCE OF SECONDARY HEMACHROMATOSIS IN PATIENT WITH THALASSEMIA MAJOR: John F. Currin. Arch. Int. Med., 93:781, (May), 1954.

Thalassemia major has been believed by many observers to represent a hereditary defect of the iron and hemaglobin metabolism. It is known that the life span of red cells in this dease is greatly reduced. Therefore, excessive amounts of iron pigments are deposited in the tissues over a period of years. The latter condition is enhanced by repeated blood transfusions and probably by increased iron absorption from the gastrointestinal tract which seems to occur in this condition. Therefore, the development of secondary hemachromatosis can be expected. So far, the latter complication has been only rarely observed. The reason is the short life expectancy of the majority of the patients. Through improvement of treatment, however, especially transfusion therapy, in recent years more patients with this disease reach adulthood. Therefore, a greater number of cases with secondary hemachromatosis will have to be expected in the future. The clinical diagnosis of this complication can only be made by liver biopsy.

H. B. Eisenstadt

CHRONIC CONGESTIVE SPLENOMEGALY WITH EARLY JAUNDICE: Robert Friedenberg and Andrew J. McQueeney. Rocky Mt. M. J. pp. 348-351, (May), 1954.

An interesting case history of a patient with hypersplenism associated with gallstone jaundice is reviewed. The 24-year old woman gave a history of remittent painless jaundice associated with marked splenomegaly for some 3½ years. It was determined that the spleen was enlarged because of thrombosis of the splenic vein. Following splenectomy the neutropenia and thrombocytopenia yielded. It is suggested that a causative relationship may exist between chronic cholecystitis, subclinical pancreatitis, and thrombosis of the splenic vein.

REGINALD B. WEILER

SEMINARS ON LIVER DISEASE, CIRRHOSIS OF THE LIVER: Charles S. Davidson. Am. J. Med., 16:863, (June), 1954.

Alcohol is responsible for the development of hepatic cirrhosis because it supplies merely calories without protein, choline and particularly methionine. Not all severe chronic alcoholics develop cirrhosis; there might be a constitutional predisposition. Not all patients with cirrhosis are symptomatic; far advanced cirrhosis might be an unsuspected autopsy finding. Marked cirrhosis might be present without abnormal liver function tests. Modern treatment of the disease may lead apparently to complete clinical recovery; however, at autopsy after death from other causes, typical cirrhotic changes will still be found. In some cases complete abstinence from alcohol and best available treatment will not prevent the progressive deterioration of the liver. The majority of cirrhotic patients do not have visible jaundice, even the total serum bilirubin is frequently normal; however, the direct one minute bilirubin is very often above normal (0.2 mg. per cent). Unfavorable prognosis is indicated by the presence of intracellular hyalin, so-called alcoholic hyalin in the biopsy specimen. Fever and marked leucocytosis may be caused by severe hepatic destruction alone without infection. Jaundice in cirrhosis is partly hemolytic, sometimes caused by hypersplenism, sometimes by the presence of a reduced red cell life span. Complete biliary obstruction with acholic stools is rare in cirrhosis but it may occur. Arteriovenous connections between hepatic artery and portal vein become more patent in cirrhosis. This contributes to the increase of portal pressure, and is the rationale of treatment with ligation of the hepatic artery. Severe portal hypertension may exist without ascites. In animals sudden obstruction of the portal veins does not cause ascites while sudden closure of the hepatic veins always produces ascites. Measurement of pressure in the abdominal collectral veins, when occluded cephalad, bears a direct relationship to the portal pressure. Hepatic coma is preceded by delirium, flapping, tremor of hands and body and paroxysmal bilateral high voltage 1½ to 3 per second in the EEG. The usual liver function tests are not helpful in the diagnosis of impending coma; however, elevated blood ammonia and oliguria not depending on salt and fluid intake are usually present at this time.

H. B. EISENSTADT

EFFECT OF PHYSICAL ACTIVITY ON RECOVERY FROM HEPATITIS: Robert S. Nelson, Helmuth Sprinz, James W. Colbert, Jr., Frank P. Cantrell, W. Paul Havens, Jr. and Margorie Knowlton. Am. J. Med., 16:780, (June), 1954.

Inadequate rest, poor diet and excessive indulgence in alcohol are considered to predispose to the development of posthepatitic sequelae. Therefore, 80 soldiers with hepatitis were re-examined 2-3 years after termination of the acute stage of their disease for residual damage. All men were apparently in good health and able to do full duty. Mild abnormalities were present in the majority of the cases. There was, however, no correlation between abnormal symptoms, signs, liver function tests, and biopsy specimens. No case justified the diagnosis of a chronic hepatitis, especially as histological examination failed to reveal any scarring of the liver. There was no evidence that activity in excess of that usually prescribed during the acute stage prolonged the course or inhibited sustained recovery. In addition, nothing suggested that indulgence in alcohol, intercurrent infection and dietary indiscretions had any untoward effect on the final outcome.

H. B. EISENSTADT

BOOK REVIEWS FOR GASTROENTEROLOGISTS

PEPTIC ULCER: C. F. W. Illingsworth, C.B.E., M.D., Regius Professor of Surgery, University of Glasgow. 287 pages, illustrated in black and color. E. & S. Livingstone, Ltd., Edinburgh and London, 1953. Williams and Wilkins Co., Baltimore, Md., 1954 Price $8.50.

This book contains 21 chapters dealing with ulcer, its diagnosis, medical and surgical treatment. In chapter one, the author discusses the discovery and recognition of hydrochloric acid as secreted by the stomach. He calls attention to observation, antedating Beaumont, by Corvisart and Leroux, of a woman who developed a spontaneous gastric fistula after an injury through which they made meticulous examination of the digestive processes. More recent additions to gastric functions have been described by Wolf and Wolff and colleagues.

Etiology, diagnosis and treatment are adequately discussed. Beautiful color plates illustrate the gastroscopic findings in ulcer and cancer of the stomach.

Medical and surgical treatment, including indications for operation and complications following operation are described.

Peptic ulcer of the esophagus and Meckel's diverticulum add greatly to the value of this well printed and illustrated volume.

It is highly recommended as a valuable addition to the physician's library.

THE BOOK OF HEALTH: Randolph Lee Clark, Jr., B.S., M.D., Professor of Surgery, Director of the M.D. Anderson Hospital for Cancer Research, Houston, Texas and Russell W. Cumley, B.A., M.A., Ph.D., Director, Editorial Dept., University of Texas M.D. Anderson Hospital, Professor of Medical Journalism, University of Texas Postgraduate School of Medicine, Houston, Texas. 1st Edition. 770 pages, beautifully printed with 1,400 splendid black and white and color illustrations. Elsevier Press, Inc., New York, N. Y., 1953. Price $12.50.

More than 300 physicians, scientists, writers, librarians, artists and photographers helped to prepare this voluminous book on health problems. A large array of physicians and others make up the advisory board, while the editorial board, in two columns per page, is spread over seven pages.

Twenty-eight chapters dealing with the beginning of life of the individual discusses everything that one wants to know regarding health and sickness, common and rare diseases, sanitation, medicine and the law, medical history, medical profession and its co-workers, the nurse, the hospital, etc.

The volume is printed on heavy coated paper, expertly and expensively illustrated and beautifully bound. Not only the laity but the physician also will find many interesting and useful facts in these pages.

AN INTRODUCTION TO BACTERIAL PHYSIOLOGY: Evelyn L. Oginsky and Wayne W. Unbreit, Merck Institute of Therapeutic Research. 416 pages, illustrated with 94 figures, 6 drawings and 18 tables. W. H. Freeman & Co., San Francisco, Calif., 1954. Price—text $6.00; trade $7.25.

The well written chapters dealing with bacterial physiology are interesting to the research scientist but are rather too much for the general practitioner.

In the preface, the authors state that they designed this volume for a course at the undergraduate level. The reviewer is happy that he is not the undergraduate student, because it seems to him one must have more than a brilliant mind to be able to absorb and evaluate this type of work.

Not to detract from the ambitious and scientific efforts of the authors, they should be further encouraged in their research and advanced studies.

STONE IN THE URINARY TRACT: H. P. Winsburg-White, M.B., Ch.B., F.R.C.S., Ed, F.R.C.S. Engl. 2nd Edition. 342 pages, illustrated in black and white and color. The C. V. Mosby Co., St. Louis, Mo., 1954. Price $16.00.

There are 21 chapters dealing with urinary calculi including calculi in children.

The nonspecialist physician may find interesting data regarding this painful disease, while the urinary specialist will be delighted with the extensive discussion as to etiology, symptomatology and treatment.

The illustrations are beyond criticism and the explanatory text is well written and to the point. This alone will make the book valuable to the many physicians who are interested in the medical and/or surgical treatment of urinary calculi.

DIE CHIRURGIE DER BAUCHSPEICHELDRUSE: Prof. Dr. Gerhard Jorons, Physician-in-Chief of the City Hospital, Arnstadt/Thiringen with an introduction by Prof. Dr. N. Guleke. 144 pages, 34 illustrations, an alphabetic reference and cross index. Walter de Gruyer & Co., Berlin W.35, Germany, 1954. Price DM 19.80.

This monograph is divided into two main parts with several subdivisions. The first part deals with the embryology, anatomy, etc., of the pancreas. Further on, the external and internal secretions and functions of the pancreas are discussed.

In the second section, the author describes the symptoms and findings in diabetes and hyperinsulinism, the medical and surgical aspects of pancreatic involvement including necrosis, cysts and benign and malignant affections.

It is a well written and illustrated little volume but adding little to our present knowledge of the pancreas.

CANCER—DIAGNOSIS, TREATMENT AND PROGNOSIS: Lauren V. Ackerman, M.D., Professor of Surgical Pathology, Washington Univ., School of Medicine, St., Louis, Mo. and Juan A de Regato, M.D., Director, Penrose Cancer Hospital, Colorado Springs, Colo. 2nd Edition. 1,201 pages, 702 illustrations, 23 in color. The C. V. Mosby Co., St. Louis, Mo., 1954. Price $22.50.

Here is a book that every physician should have. It is easily read and has superb illustrations. It discusses diagnosis, treatment, results and research. No organ in the body is omitted as the reader may note. Chapter seven—Cancer of the Respiratory System and Upper Digestive Tract, occupies 277 pages. Cancer of the Digestive Tract is 210 pages. These two chapters are the largest, most descriptive and explanatory chapters in the volume dealing with neoplastic conditions.

It is highly recommended as a worthwhile convenient and reference book on neoplastic diseases.

CARCINOMA OF THE COLON: Leland S. McKittrick, M.D., Clinical Professor of Surgery, Harvard Medical School and Frank C. Wheelock, Jr., M.D., Assistant in Surgery, Massachusetts General Hospital, Boston, Mass. 94 pages illustrated. Charles C. Thomas, Springfield, Ill., 1954. Price $3.25.

Carcinoma of the Colon is one of the American Lecture Series edited by Lester R. Dragstedt, M.D. Although this text consists of 94 pages, it covers the subject quite well. It is easy to read and readily assimilated.

The internist as well as the surgeon will find many useful data and therefore, it is recommended as a valuable adjunct for the physician's desk.

PRIMER OF ALLERGY: Warren T. Vaughan, M.S., M.D. Revised by J. Harvey Black, M.D. 191 pages illustrated. The C. V. Mosby Co., St. Louis, Mo., 1954. Price $4.25.

This little volume is now in its 4th edition and brought up to date since Dr. Vaughan's death. It is useful both to the physician and the lay reader. Many obscure questions as to diet, injections for desensitization, cosmetics and disinfectants are answered satisfactorily.

General orders and directions to the patient are clearly outlined and should be read by those suffering with allergy.

The Primer of Allergy is highly recommended for the general practitioner, who in turn will greatly benefit his allergic patients by recommending the purchase of this explicit primer.

PRACTICE OF ALLERGY: Warren T. Vaughan, M.D. Revised by J. Harvey Black, M.D. Third Edition. 1,164 pages well illustrated. The C. V. Mosby Co., St. Louis, Mo., 1954. Price $21.00.

This well written and illustrated volume by the late Dr. Vaughan is thoroughly revised and brought up to date by Dr. Black. The book is divided into 16 parts and 76 chapters covering the entire question of allergy. The reviewer, who is familiar with the previous two volumes, is amazed with the amount of new material and extensive bibliography presented, making this book the outstanding text on allergy.

No further elucidation or discussion can add to the value of this volume unless the physician will buy and read it; then only will he agree with the reviewer that it is a worthwhile addition to his library.

HUMAN BIOCHEMISTRY: Israel S. Kleiner, Ph.D., Professor of Biochemistry and Director of the Department of Biochemistry, New York Medical College, Flower and Fifth Avenue Hospital, New York, N. Y. Fourth Edition. 746 pages, illustrations in black and white and color. The C. V. Mosby Co., St. Louis, Mo., 1954. Price $7.50.

The 4th edition of Dr. Kleiner's Human Biochemistry has been brought up to date and many parts have been rewritten or elaborated upon. Students in medical colleges, graduate students preparing for higher degrees in medicine or teaching will find this edition an excellent guide.

Men who are preparing for medical licenses or basic science examinations will be greatly benefited by studying the text.

The reviewer highly recommends Human Biochemistry as a useful and valuable monograph.

KLINISCHE DARMBAKTERIOLOGIE FUR DER ARZTLICHE PRAXIS: Prof. Dr. med. et phil. Tr. Baumgartel, Munchen. 130 pages. Georg Thieme Verlag, Stuttgart, 1954. Price DM 14.40 ($3.40).

Unfortunately this well prepared essay on the intestinal flora and therapy is written in a language which many American physicians cannot read. There are many excellent ideas as to diagnosis and suggestions for treatment which the reviewer finds worthwhile.

Physicians who do read German and are interested in gastrointestinal diagnosis and treatment should without fail add this small monograph to their library.

DIET MANUAL OF THE JEWISH HOSPITAL OF BROOKLYN, N. Y.: Edited by Charles Solomon, M.D., F.A.C.P.; Nanette D. Robertson, B.S., M.A. and Miriam C. Teknesky, B.S. 87 pages, American Book. Stratford Press, Inc., New York, N. Y., 1954. Price $2.50.

The dietary manual is a compilation of diets used by the medical staff of the Jewish Hospital of Brooklyn.

In addition to the editors, members of the visiting staff representing some of the specialties, pediatrics, allergy and gastroenterology, help to make a workable manual of diets.

It is recommended highly both to the specialist and the general practitioner.

DISEASES OF THE LIVER: Mitchell A. Spellberg, M.D., F.A.C.P., Associate Professor of Clinical Medicine, University of Illinois School of Medicine. 646 pages, illustrated. Grune and Stratton, New York, N. Y., 1954. Price $16.50.

In this volume, the reader will find information relating to the anatomy and physiology of the liver and many illustrative photographs, drawings, tables and diagrams. At the end of chapters, there are summaries which are useful aids in reviewing and in teaching.

Needle biopsies, differential diagnosis, bibliographies and cross-index make Dr. Spellberg's book an excellent publication and it is highly recommended by the reviewer.

THE HEPATIC CIRCULATION AND PORTAL HYPERTENSION: Charles G. Child, III, M.D., Professor of Surgery, Tufts College Medical School; Chairman, Department of Surgery, New England Center Hospital. From the Department of Surgery and the Laboratory of Surgical Research of the New York Hospital-Cornell Medical Center In collaboration with Ward D. O'Sullivan, M.D., Mary Ann Payne, M.D., George R. Holswade, M.D., David Barr, M.D., etc. 444 pages with 132 figures. W. B. Saunders Co., Philadelphia, Pa., 1954. Price $12.00.

Although hepatic circulation and portal hypertension is a timely subject, many physicians are not very familiar with the various ramifications of this condition. The reviewer recommends that all physicians, medical students, residents and interns read the book carefully and they will be delighted with the wealth of information within its pages.

An extensive bibliography and cross-index are special features for which the authors and publishers should be highly commended.

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1. ROGERS, M.P., AND GRAY, G.L.: AM. J. DIGEST. DIS. 19:180 (JUNE) 1952.

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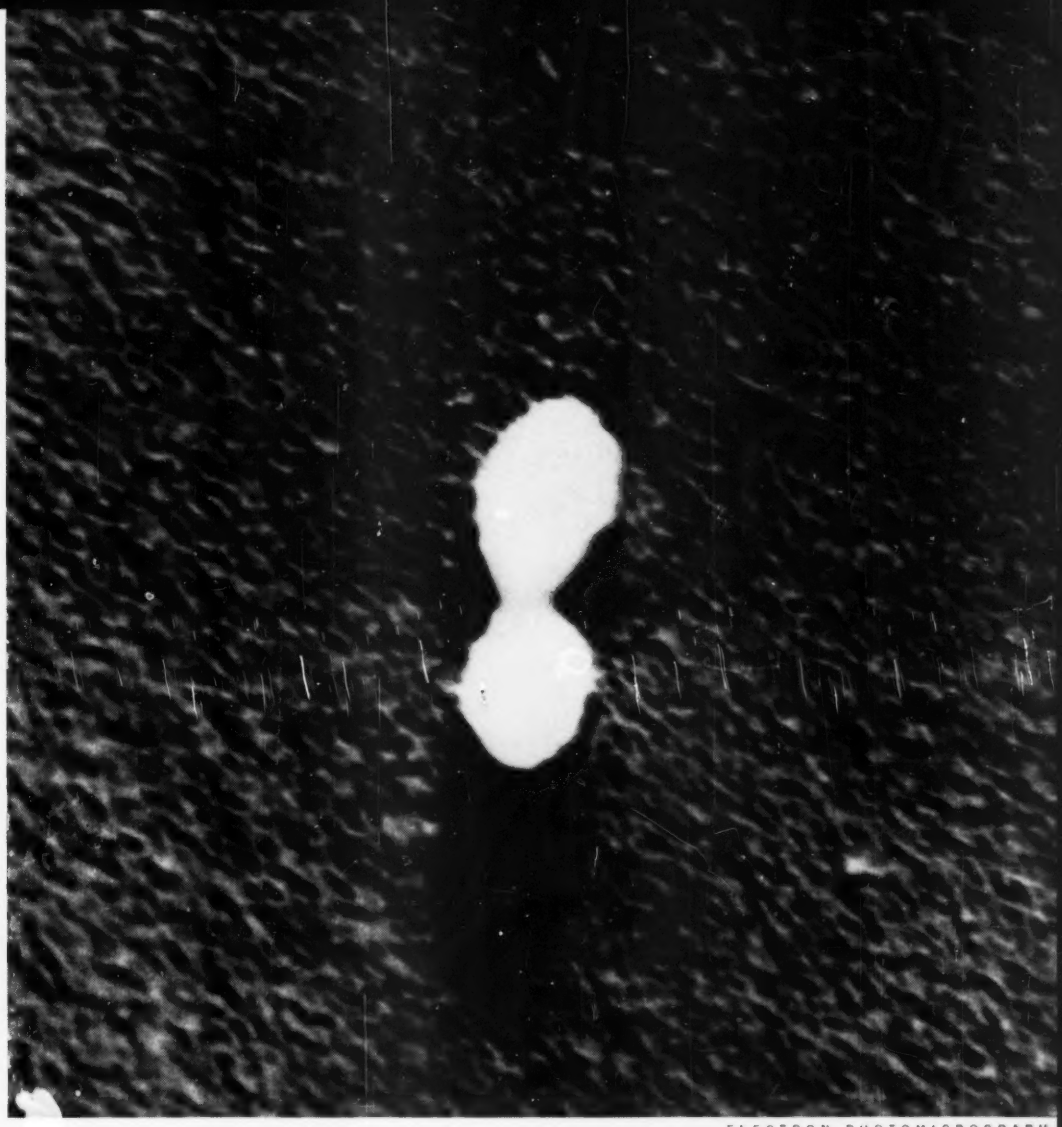

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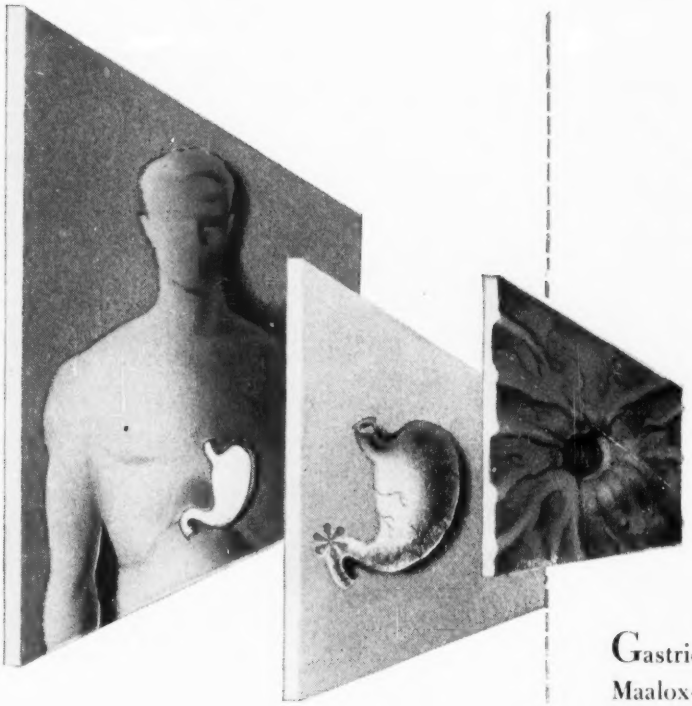

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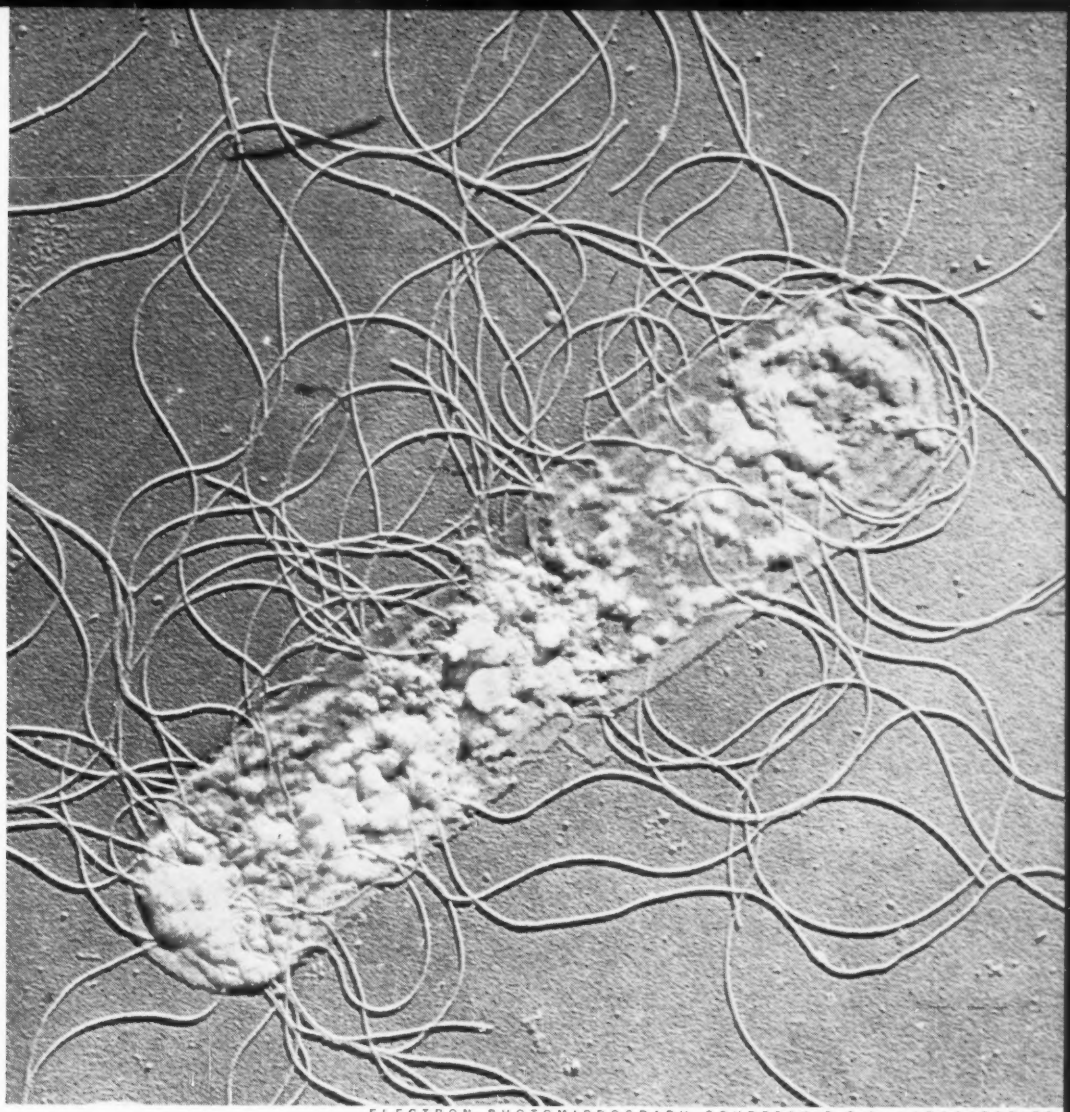

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1. Rossett, N. E., and others: Ann. Int. Med. 36:98 (*Jan.*) *1952*

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